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EDITORIAL

SURGICAL TREATMENT OF TUBERCULOSIS OF THE LUNG

An American visitor to the Brompton Hospital remarked that the treatment of pulmonary tuberculosis is 100 per cent. surgical. Thoracic surgery has made such strides in recent years that we should be inclined to agree with him if he would omit the second " 0 " and call it 10 per cent.

In our last issue we published an article by Dr. Gravesen on phrenic evulsion, and in this number will be found an article by Mr. Morriston Davies dealing with thoracoplasty and one by Drs. Haberlin and Hudson on extra-pleural plombage.

These authors have all written with restraint, and not suggested that surgery is any more than an aid in the treatment of pulmonary tuberculosis.

Mr. J. E. H. Roberts, though equally modest in his claims, has given in the recent Lettsomian Lectures a very clear outline of the great advances which have been made in this branch of surgery.

Lobectomy has not yet been performed for tuberculosis, but the operation is becoming so safe and successful in other conditions that it may soon be possible to excise certain tuberculous lesions of the lung.

At present it would seem that such treatment could rarely if ever be wise, because the real value of surgery in the treatment of tuberculosis has so far been merely as a mechanical method of giving rest to the diseased part. Operations for other purposes in all forms of tuberculosis often lead to disastrous results.

Dr. Jaquerod's paper should be read carefully by the surgeon as well as the physician, for he presents a very good case for those who advocate conservative methods of treatment before resorting to artificial pneumothorax or surgical means of collapsing the lung. Certainly a patient often gets into that state when surgery, and only surgery, can offer a chance of recovery; but it is the duty of the physician to prevent him from getting into that state, and, as Dr. Jaquerod points out, when once one embarks on surgical procedures, one may be faced with the necessity of further operations each more serious than the last.

PROBLEMS IN APPLIED MEDICINE

WHY THE PULMONARY FORM OF TUBERCULOSIS IS COMMON IN ADULTS AND THE NON-PULMONARY FORM IN CHILDREN

By P. F. ARMAND-DELILLE,
M.D.
De l'Hôpital des Enfants Malades, Paris.

Every pathologist, and any doctor who has a large practice, know that many babies who have been in contact with tuberculous adults die from generalised miliary tuberculosis; also that in infants who have been exposed to contagion in the same manner death is due either to tuberculous meningitis or to generalised or lung miliary tuberculosis. Death from these causes is less frequent after infancy, but local forms of tuberculosis, such as glands, joints, bone, and sometimes peritonitis or pleurisy, occurs. By the time puberty is reached pulmonary tuberculosis is more frequent and, in adults, it is the most common manifestation of tuberculosis.

There is, however, a certain difference between the pathologist's and the practitioner's knowledge, because very often at the autopsy of children who succumbed to some other disease the pathologist discovers the presence in the lung of a more or less calcified or caseated tuberculous nodule, and also the presence of tuberculous lymph glands in the mediastinum. This was first described by Parrot, and later by Küss, as " chancre d'inoculation avec adénopathie," and by Albrecht and Ghon as " Primär Komplex." Practically, this indication is of no value, because the primary infection seldom produces a caseous pneumonia or cavity. As a rule such early manifestations heal or remain inactive until they produce at the time of puberty a reinfection of the lung.

For a long time it was impossible to understand this difference between adults and young people. It is only during the last thirty years that a series of experiments has enabled us to explain, at least partially, these conditions.

The discovery of allergy by von Pirquet has brought a great change in our conception of the tuberculous infection. By demonstrating that the introduction of tuberculin into a scratch of the skin provokes no reaction in a sound organism, but a local inflammation with redness and œdema in a previously infected organism, he showed that the body becomes modified by the first penetration of tubercle bacilli.

We know that in 1891 Koch described an experiment whereby he gave a second hypodermic injection of virulent living bacilli to a guinea-pig which had been previously infected in the same manner more than fifteen days before.

If one injects virulent tubercle bacilli under the skin of a guinea-pig, ten days after the injection the animal develops a small local abscess, and after a few more days a swelling of the corresponding lymph glands, in the axillary and crural territory. Later on, the infection reaches the tracheo-bronchial glands, and after two or three months the animal dies from generalisation of the infection.

But if about fifteen or twenty days after a guinea-pig has been inoculated a dose of virulent bacilli is injected, one can observe within about two days the presence of a local nodule, with congestion and redness of the skin. A few days later an abscess appears, which soon opens spontaneously. After the abscess is drained there remains an ulceration in which bacilli may be found for a certain length of time. But reaction of the corresponding lymph glands is never observed in those cases. The local reaction corresponds to the positive Pirquet reaction for tuberculin—that is to say, the first injection has conferred new defensive properties upon the organism, so that the infection is limited to the point of penetration of the pathogenic agent, with necrotic reaction, and the surrounding lymphatic or vascular territories are not involved.

In such conditions it is easy to understand that the hæmoptysis or the hæmaturia are only the symptoms of the reaction of the lung or the kidney after secondary localisation of the bacilli on these organs. It also explains why one never finds hæmoptysis in young children—that is, at the primary stage of infection, when allergy is absent or very faint. In other children or young adults, when allergy is only partially established, one may observe large congestive reactions, such as induration and redness of the skin in the neighbourhood of small tuberculous emboli, which is characteristic of *erythema nodosum*, or the large infiltrations that one sees in splenopneumonia.

If allergy is fully established—that is to say, if the resistance of the organism to tuberculosis infection is really increased—the patient is able to offer an exclusively local reaction to any inoculation of tuberculosis. In these

conditions, why is pulmonary tuberculosis more common in adults, while the non-pulmonary form is more often found in children?

Experiments are misleading, because the reactions obtained with animals are very different from those obtained with humans. The life of the animal is shorter, its growth more rapid; only apes and chimpanzees can stand the comparison, and we know that even these animals have a special sensibility to the tubercle bacillus, that they react with more sensitiveness, as is the case with coloured patients, who, like young children, show a complete lack of resistance to tuberculous infection.

We shall study later the question of tuberculosis among negroes of non-infected regions, which Borrel had the opportunity of observing in the French black troops during the Great War.

Though many points still remain obscure, we can say that two principal conditions account for the difference in localisation and form in adults and children.

General Conditions.

The adult has allergy, and this tends to produce localisation.

The child, and especially the infant, has primary infection which becomes generalised, and he dies from disseminated tuberculosis or meningitis.

According to my statistics, 60 per cent. of the infants and children in contact with tuberculous parents present a characteristic tuberculous evolution before the death of the tuberculous parent, and two-thirds of them (that is, 40 per cent.) die from acute forms—miliary or tuberculous meningitis; only one-third of these (60 per cent. infected) present chronic localisations. Forty per cent. seem to be clinically sound, but they have a positive Pirquet, and many present small shadows in the lungs or in the hilus from an inactive primary affection which may wake up later, at the time of puberty and adolescence, and be the beginning of a chronic pulmonary tuberculosis.

In adults the lung seems to be the organ that offers less resistance to tuberculosis, perhaps because its capillary system constitutes a kind of screen for all the tubercle bacilli carried by the blood stream. It seems that, with the exception of the kidneys and genital organs, most of the tissues have acquired a special resistance against the infection.

On the contrary, in the case of children whose organs are still growing, the organs which are developing the most rapidly appear to be specially *locus minoris resistentiæ* for the bacillus.

We know now, with the help of radiography, that the generalisation which is the rule in primary infection is not always fatal; besides the cases of "granulies froides," described by Burnand and Sayé, there are many cases when a discrete generalisation may appear. The majority of these

disseminated miliary tubercles may heal spontaneously; but a few of them, developing in *locus minoris resistentiæ*, may spread the disease in their surroundings, as is the case in meningitis, or may give a local caseation, as in bone or joints tuberculosis.

If the child develops only one or a few local lesions it probably means that he has acquired a certain degree of resistance. As a fact, small infants do die from generalised miliary or meningitis, and it is mostly in young children or adolescents that one observes the local forms of tuberculosis—Pott's disease, tuberculosis of the cervical glands, of the kidneys or liver, pleurisy with effusion or spleno-pneumonia, which is also a curable form of lung tuberculosis. As the child gets older he (like the adult) more often presents reinfection (mostly auto-reinfection)—pulmonary tuberculosis with ulcero-caseous form.

Another point to be considered is what has been called the "Max Schultze's law": if some traumatism is present, the microbes injected or circulating in the blood stream will localise on the wounded point. Very often, indeed, parents declare a fall to have been the cause of hip tuberculosis or Pott's disease. Recently, in a paper which I read in London at the meeting of the Tuberculosis Association, I discussed the question of tuberculous reinfection of the lungs. It really seems that localisation on the lungs is seldom due to recent contact with germ carriers, but that, on the contrary, it follows the reactivation of an old infection of the lungs contracted many years before. In such cases the patient had had—as an infant—a primary infection which spread small foci in the lungs; the foci healed spontaneously, seemed to calcify, and remained dormant for many years, then woke up when the general resistance of the organism weakened on account of the rapid growth in adolescence. We know that at that period of life girls undergo a rapid decalcification which is particularly observed on the teeth. With boys the conditions are a little different, because puberty comes slowly, but—especially in the working class, or among young students who have to furnish constant effort in the preparation of college or university examinations—the reactivation of the old infection is frequent and may provoke severe forms of pulmonary tuberculosis. We know that, on the contrary, the older an adult grows the more he becomes resistant to tuberculosis. A great number of old people complaining of a little cough or of slight bronchitis are really germ carriers presenting a more or less developed cavity in the lungs. Such patients may live for years and reach a very advanced age. They look quite healthy, but are a terrible danger to their grand-children; and if they live in the same house, many of the children die from miliary or from tuberculous meningitis. These conditions are observed among adults living in the big or small cities of our civilised countries of Europe

and America. But if an adult who has never been in contact with tuberculous surroundings comes to live in our countries, he reacts like an infant and not like an adult.

Borrel, of the Pasteur Institute, had the opportunity of taking care of a hospital of coloured people at Marseilles during the second part of the Great War, and to observe a great number of sick soldiers. Many of these men developed miliary or caseous pneumonia, because they had no resistance against the bacillus; others developed big caseous glands; but very few presented joints or bone tuberculosis, because, being adults, they had no *locus minoris resistentiæ* in these parts of the body.

We must also remember that pulmonary tuberculosis is not so infrequent in infants or small children. In our book published in collaboration with Dr. Lestocquoy we have shown that extensive caseation of one lung is not rare at that period of life, though miliary or tuberculous meningitis is much more frequent. As a rule, if the young patient does not seem to develop pulmonary tuberculosis, it is because this slow form has no time to develop in the infant, who, owing to absence of resistance and allergy, very soon develops generalisation of the disease.

The above considerations may help us to understand why the pulmonary form is common in adults and the non-pulmonary form in children, and, moreover, that primary infection, as Ranke considered it, nearly always gives a local " Primär Komplex " which may be followed by generalisation, or remain (locally) latent until it provokes reinfection with, as a rule, localisation confined to the lungs, as these organs seem to offer less resistance to the infection.

GENERAL ARTICLES

A STUDY OF TUBERCULOUS DISEASE IN INFANCY AND CHILDHOOD, WITH PARTICULAR REFERENCE TO THE PRIMARY SITES OF INFECTION

By JOHN W. S. BLACKLOCK
M.D. (GLAS.)

(From the Department of Pathology of the University and Royal Hospital for Sick Children, Glasgow)

THERE is no doubt that the type of tuberculosis and type of bacillary infection depend largely on the geographical distribution of the disease. Had Koch lived in Scotland, where primary abdominal tuberculosis due to the bovine type of tubercle bacillus is common, it is certain that he would not have made the statement that this bacillus was relatively innocuous to the human subject—a statement which led to intensive world-wide investigation.

It is thus of much importance that there should be a free exchange of opinion by the workers of various countries regarding the pathology and bacteriology of tuberculosis in the early years of life. By this means the solution of many obscure problems in tuberculosis—*e.g.*, the differences in the types of lesions and bacillary infections—may be reached.

In 1932 we published an account of the results of an investigation of tuberculous disease in children which dealt partly with the types of lesions and strains of bacilli found in a series of cases examined postmortem. On account of many points which were at variance with those recorded by Continental and American workers, it seemed of importance to continue this investigation, and the results of these further observations, together with those previously recorded, are described in this paper.

Over a period of ten years we have examined every subject that has come to autopsy at the Royal Hospital for Sick Children, Glasgow, for

evidence of tuberculous infection. In all, we have studied a consecutive series of 2,500 cases, the ages of these children varying from a day up to thirteen years. In this large group of unselected cases there were 372 (14·9 per cent.) with tuberculous lesions. Naturally, one must not regard the findings recorded here as applicable to the child population as a whole. The subjects belonged to the poor and working classes, and the diseases, either acute or chronic, from which they were suffering may have had an unfavourable influence on any tuberculous lesion present. Furthermore, there was the possibility of selection outside the hospital, some of the more chronic cases of tuberculous disease, especially in older children, being sent by their own doctors, in co-operation with the public health authorities, to special institutions set apart for the treatment of this disease.

The Age and Sex Incidence of Tuberculous Lesions.

The subjects coming to autopsy and those in which tuberculous lesions were found are arranged according to age-groups in Table I. The lowest

TABLE I.

INCIDENCE OF CASES WITH TUBERCULOUS LESIONS ACCORDING TO AGE-GROUPS IN 2,500 CONSECUTIVE AUTOPSIES.

Age-Group.	*Total Autopsies.*		*Tuberculous Cases.*			
			Number.		*Percentage.*	
0 to 3 months	790	1,688 in 1st year	6	110	0·76	6·52
3 to 6 ,, ...	433		34		7·85	
6 to 9 ,, ...	274		38		13·87	
9 to 12 ,, ...	191		32		16·75	
12 to 18 ,, ...	192	314 in 2nd year	50	86	26·04	27·4
18 to 24 ,, ...	122		36		29·5	
2 to 3 years	124		53		42·7	
3 to 6 ,, ...	198		70		35·4	
6 to 9 ,, ...	127		36		28·3	
9 to 13 ,, ...	49		17		34·7	
Total	2,500		372		14·9	

percentage (0·76) of cases with evidence of tuberculosis was found in the first three months of life. Thereafter there was a steady increase up to the peak (42·7 per cent.) in the third year, with a decline in each of the succeeding three-year periods up to thirteen years. Under three years of age, the percentage incidence of cases with tuberculous lesions to the total autopsies was 11·7 and 32·9 per cent. between three and thirteen years. It is interesting to note that throughout the whole of childhood the incidence of tuberculous lesions in this series was always greater in girls than in boys (Table II.).

TABLE II.

INCIDENCE OF CASES WITH TUBERCULOUS LESIONS ACCORDING TO SEX AND AGE-GROUP IN 2,500 CONSECUTIVE AUTOPSIES

			0 *to* 1 *Year.*	1+*to* 2 *Years.*	2+*to* 3 *Years.*	3+*to* 6 *Years.*	6+*to* 9 *Years.*	9+*to* 13 *Years.*	*Total.*
BOYS	Total autopsies		1,048	185	70	101	73	29	1,506
	Tuberculous cases	Number	64	45	29	25	18	9	190
		Percentage	6·1	24·3	41·4	24·8	24·7	31·0	12·6
GIRLS	Total autopsies		640	129	54	97	54	20	994
	Tuberculous cases	Number	46	41	24	45	18	8	182
		Percentage	7·2	31·8	44·4	46·4	33·3	40·0	18·3

The cause of this sex difference cannot be explained in the early years of life, though in the later years of childhood girls belonging to the poorer classes probably do not lead such active outdoor lives as their brothers. Consequently, they are more exposed to any tuberculous infection which may exist in the home circle, and, further, on account of the indoor life, are probably less resistant to this infection.

TABLE III.A.

THE RESULTS OF 834 MANTOUX TESTS ON CHILDREN IN THE PRESENT SERIES COMING TO AUTOPSY.

Age (Years).	*Non-tuberculous.*		*Tuberculous.*		*Per Cent. Positive.*
	Negative.	*Positive.*	*Negative.*	*Positive.*	
0 to 1	498	6	12	34	7·3
1 to 2	82	2	8	34	28·6
2 to 3	16	—	8	19	44·2
3 to 6	22	—	11	26	44·1
6 to 9	21	—	7	12	30·0
9 to 13	7	2	1	6	50·0
Total ..	646	10	47	131	16·9

In order to be certain that we were not missing any tuberculous lesion, all the cases that came to autopsy with positive tuberculin tests were most carefully studied in order to find the lesion which had caused the super-sensitiveness to tuberculin. In 834 cases which came to necropsy, accurate Mantoux tests had been performed with dilutions of old tuberculin varying from 1 in 5,000 to 1 in 10. In 10 only, where the Mantoux test was positive, no tuberculous lesions were found. Of these 10 cases, 8 were in the first year of life (Table III.A). It is interesting to observe that 47 (26·4 per cent.)

of the 178 tuberculous Mantoux-tested cases failed to react to the dilution of tuberculin used. Of course, many of these children died a few days after admission, so that any extensive investigation with concentrations of tuberculin higher than that used for the initial test could not be undertaken.

TABLE III.B.

THE RESULTS OF 1,199 VON PIRQUET TESTS ON CHILDREN IN THE PRESENT SERIES COMING TO AUTOPSY.

Age (Years).	*Non-tuberculous.*		*Tuberculous.*		*Per Cent. Positive.*
	Negative.	*Positive.*	*Negative.*	*Positive.*	
0 to 1	710	10	35	51	7·6
1 to 2	118	5	21	44	26·1
2 to 3	16	—	9	27	51·9
3 to 6	32	1	14	29	26·3
6 to 9	30	4	8	16	34·5
9 to 13	10	4	3	2	31·6
Total ..	916	24	90	169	16·1

In a series of 1,199 von Pirquet tests (Table III.B) made on children, all of whom were examined post mortem, no tuberculous lesion was found in 24 who gave positive results, while 90 (34·8 per cent.) of the 259 Pirquet-tested tuberculous children gave negative results. Some children were tested both with the Mantoux and Pirquet tests, so that there was some overlapping of the two series. It is interesting to observe that the percentage of tuberculous cases giving negative von Pirquet tests is higher than those giving negative Mantoux reactions. This confirms the observations of Hart (1932) regarding the greater accuracy of the Mantoux than of the von Pirquet test. Further discussion on the results of these tuberculin tests is not possible here, as the work is not yet completed and will be published in full at a later date. It is apparent, however, that the percentage of tuberculin-positive cases corresponds fairly closely with the percentage incidence of the lesions found postmortem in the various age-groups (Table I.). Accordingly, the figures given for the incidence of tuberculous lesions found at autopsy in this series of children can be regarded as correct.

Distribution of the Primary Sites of Infection.

Early recognition of the primary sites of infection is important to the clinician in his attempt to arrest the disease in incipient cases, and so prevent the secondary and usually fatal generalisation. Accordingly, in this

series the primary sites of infection were determined as accurately as possible, and these were as follows:

Thoracic	239 (63·4 per cent.).
Abdominal	123 (32·4 per cent.).
Cervical	8 (2·1 per cent.).
Not found	7 (2·1 per cent.).

In 5 cases there were double primary sites of infection, one in the thorax and the other in the abdomen. Thus the primary site of the tuberculous disease was commonest in the thoracic organs (lungs or lymphatic glands, or both), next in the abdomen (intestines, mesenteric glands, or both), then the cervical glands, and lastly, in a few cases, the primary site of infection was not discovered.

Primary Thoracic Tuberculosis.

Of the 2,500 subjects studied, 2,126 were under three years, and of these 161 (7·6 per cent.) had primary tuberculous lesions in the thoracic organs. Similar lesions were found in 78 (20·9 per cent.) of the 374 cases between three and thirteen years. Thus, while there was a greater number of cases of primary thoracic tuberculosis under three years in this series, yet the relative incidence of this type of tuberculosis to the number of autopsies was greater in children over three than in those under. Only one (0·6 per cent.) of the children under three years and seven (8·97 per cent.) of those over this age did not die as a result of the intrathoracic tuberculous disease or its secondary effects (Table VI.). Accordingly, we must conclude that in Scottish children intrathoracic tuberculosis is a relatively fatal disease, especially in the first three years of life. Continental and American authors, on the other hand, have reported numerous cases where the disease in the lungs and glands has become arrested, and in view of this discrepancy between these observations and our own we decided to investigate the matter as fully as possible. In support of our results was the fact that in our experience with adult subjects examined postmortem the presence of healed tuberculous glands in the thorax was uncommon. Professor Sir Robert Muir has also informed us that, from an extensive experience of autopsies in Glasgow, the presence in adults of calcified thoracic glands indicating healed tuberculosis is relatively rare. Further, as we have already shown, there was in this series only a very small number of children with positive Mantoux tests in whom no tuberculous lesions were found at autopsy, so that probably only a very small number of cases of early tuberculosis or small healed lesions were missed. In a further uncompleted series of tuberculin tests in *clinically* non-tuberculous children under three years who have not died, only 7·6 per cent. have given positive results.

In order to help further in the elucidation of this problem, we have, during the last three years, examined stomach washings for tubercle bacilli from all children admitted to the hospital with clinical and radiological evidence suggestive of pulmonary tuberculosis, and who gave positive Mantoux reactions. The results of this investigation are recorded in Table IV., from which it is evident that the majority (17 out of 25) of the cases were over three years, and that of the 8 cases under this age, 4 died of tuberculosis. In only six instances were tuberculous lesions produced in guinea-pigs inoculated with the sediment (treated either with 7·5 per cent. antiformin or 4 per cent. sodium hydroxide) obtained from these washings. On typing these strains they were all found to be human. In the 2 cases under three years in which bacilli were found, both children are still alive and well fifteen and thirteen months respectively after dismissal, and in the older child (now three years two months) the lesions are showing radiological evidence of calcium deposition. Of the two children under three in whose stomach washings no tubercle bacilli were found, one (fourteen months after dismissal) shows evidence of generalisation of the disease; the other case could not be traced. Thus in this small series the rarity of arrest of pulmonary tuberculosis in the young child was again evident.

TABLE IV.

RESULTS OF STOMACH WASHINGS IN 25 CASES WHERE PULMONARY TUBERCULOSIS WAS SUSPECTED CLINICALLY.

Result (Guinea-Pig).	*State of Child.*	*0 to 1 Year.*	*1 to 2 Years.*	*2 to 3 Years.*	*3 to 13 Years.*	*Total.*
6 positive	Alive	—	1	1	1	3
	Died	3	—	—	—	3
19 negative	Alive	—	2	—	16	18
	Died	1	—	—	—	1

Accordingly, it would appear that our statement regarding the almost invariable fatal issue resulting from thoracic tuberculosis in children in the locality studied is actually the case when the following facts are considered—viz.:

(1) The great rarity of healed pulmonary tuberculosis of infantile type at autopsy in adults belonging to Glasgow and the West of Scotland.

(2) The close agreement between the percentage incidence of positive tuberculin tests and the percentage of tuberculous lesions found at autopsy in the same series of children.

(3) The rarity in a large autopsy series and in a smaller clinical series of definite arrested pulmonary tuberculosis in children, especially in those under three years of age.

We are of the opinion, however, from a survey of the records of our department over a period of fifty years, that the severity of the disease is decreasing. Why intrathoracic tuberculosis should cause death so frequently in young Scottish children is difficult to explain. Hendrie (1933) has stated that in children in the Birmingham district much the same holds good. Is it that British children are less resistant to infection on account of racial, dietetic, or climatic factors? These are all very important points for discussion, and if a solution is possible, we might be able to reduce substantially the death-rate for tuberculosis in the first few years of life.

Types of Lesions.

We have already (1932) described fully the types of tuberculous lesions found in the lungs of children, and will only mention some additional facts here. Typical primary foci with related enlarged caseating glands were found in 213 of the 239 present thoracic cases; in the remaining 26, though the tracheo-bronchial glands were caseous, no primary lung lesion was found. The distribution of the primary lung lesions was as follows:

192	cases	had	1	primary	focus	each = 192
14	,,		2	,,	foci	,, = 28
4	,,		3	,,	,,	,, = 12
3	,,		4	,,	,,	,, = 12

In all 244 primary foci were found in 213 cases, and in the majority of subjects they were single.

A study of the naked-eye characteristics of these 244 lesions regarded as the primary site of infection in the lungs is of interest, and is given in detail in Table V., from which it is evident that it was in the early years of life that the more acute type of primary lesion was found. Further, in lesions in children under six months there was no evidence of healing as judged by naked-eye examination, while after that age the incidence of attempted arrest—*e.g.*, surrounding fibrosis, calcification, or both—increased *pari passu* with the age of the child. Partial calcification of the lesions was, however, a rarity, occurring in only 20 of the 244 foci. In subjects under three years the incidence of healing was 21·9 per cent., while in those between three and thirteen years it was 62·7 per cent. Accordingly, from these facts it is evident that in the case of the young Scottish child the tuberculous lung lesion at an early age tends to be progressive, and thus fatal, whereas later in childhood there is a very much better chance of arrest of the disease.

In these 213 cases, however, the primary foci were the only lung lesions in 17, in 8 of which there was pleural involvement. In all the others there was extension of the tuberculosis to other parts of the lungs—viz., miliary tubercles in 36, subacute miliary tubercles in 56, a combination of miliary tubercles and small broncho-pneumonic tubercles in 80, broncho-pneumonic tuberculosis in 19, and chronic peribronchial lymphatic tubercles in 5. Thus in nearly all our thoracic cases, in addition to the primary focus, there were other secondary tuberculous lesions in both lungs.

TABLE V.

AGE DISTRIBUTION OF PRIMARY LUNG FOCI ARRANGED ACCORDING TO MACROSCOPIC CHARACTERS.

Type of Lesion.	0 *to* 6 *Months.*	6 *to* 12 *Months.*	1 *to* 2 *Years.*	2 *to* 3 *Years.*	3 *to* 6 *Years.*	6 *to* 13 *Years.*	*Total.*
Caseous	19	15	25	15	15	7	96
Cavity, caseous walls ..	15	28	10	5	2	4	64
Total showing *no* healing	34	43	35	20	17	11	160
Caseous + fibrosis	—	1	16	10	6	14	47
Caseous + early calcification + fibrosis	—	—	1	2	8	9	20
Cavity, caseous walls + fibrosis	—	—	5	2	7	3	17
Total showing healing..	—	1	22	14	21	26	84
Per cent. showing healing ..	—	2·3	38·6	41·2	55·3	70·3	
			21·9		62·7		

As regards the extra-pulmonary lesions, we have already stated that in the 213 cases with primary lung lesions and in the 26 in which these were not discovered the tracheo-bronchial glands were enlarged and caseous. In general, the younger the child the greater was the extent and degree of glandular involvement. There had been direct extension of the lesion from the thoracic glands to the deep cervical group in 49, and to the glands in the upper part of the abdomen in 20. In 86 subjects the infected sputum had caused ulceration of the bowel from which the tuberculous disease extended to the mesenteric glands. These glandular and intestinal lesions were always more extensive in the younger children. When the cause of death is considered in these thoracic cases, it will be seen from Table VI. that of the 161 cases in children under three, only 5 (3·1 per cent.) died from the actual disease in the lung alone, while in children over three years

the corresponding percentage was 5·1. With these exceptions, in addition to one child under three years and seven over this age who died from causes other than tuberculosis, all the others showed tuberculous meningitis, tuberculomata, generalised miliary tuberculosis, or various combinations of these. Such generalisation was found in 96·3 per cent. of children in the first three years of life and in 85·9 per cent. of those between three and thirteen. Thus the bad prognosis in our primary thoracic cases was largely

TABLE VI.

CAUSE OF DEATH IN CASES OF PRIMARY THORACIC TUBERCULOSIS.

Lesions.	0 *to* 1 *Year.*	1 *to* 2 *Years.*	2 *to* 3 *Years.*	*Total* 0 *to* 3 *Years.*	3 *to* 13 *Years.*
Cerebral, generalised miliary tubercle, or both	74	50	31	155 (96·3)	67 (85·9)
Lung disease	2	3	—	5 (3·1)	4 (5·1)
Not cause of death	—	—	1	1 (0·6)	7 (9·0)
Total	76	53	32	161	78

Percentages in brackets.

due to the spread of the disease to other parts of the lungs, to generalisation by means of the blood and lymph streams, and this was slightly commoner in the younger children. If it were possible to keep the tuberculous disease localised to the lungs and related glands, the high mortality might be reduced. The great difficulty in carrying this out, however, is that once the paratracheal glands are infected the tubercle bacilli are on the threshold of the blood stream, as the efferents from these glands frequently drain directly into the great veins in the thorax, thus allowing infection to become rapidly generalised.

Types of Bacilli found in the Thoracic Cases.

In 149 cases where the primary lesion was in the thorax and in which the patient had died of tuberculosis, the infecting organism was isolated and typed both by culture and by inoculation of rabbits. Of these, 143 were human and 6 (4 per cent.) were bovine strains. The bovine percentage was 4·6 in children under three years and 2·4 in those between three and thirteen (Table VII.). Of the human strains, 105 were isolated from the lung lesions or associated tracheo-bronchial glands, 35 from the cerebro-spinal fluid of cases of tuberculous meningitis in which the primary site of infection was found post mortem in the thorax, and 3 from stomach washings of children who came to autopsy and in whom typical primary complexes

were found in the lungs. Of the 6 bovine strains, 3 were isolated from the tracheo-bronchial glands, 3 from the cerebro-spinal fluid of meningitis cases which came to autopsy and in which typical primary lesions were found in the lungs. Human strains were obtained from 5 cases which did not die as a result of the lung lesions; in 3 of these the bacilli were isolated

TABLE VII.

TYPES OF BACILLI ISOLATED FROM CASES OF PRIMARY THORACIC TUBERCULOSIS.

		0 *to* 1 *Year.*	1 *to* 2 *Years.*	2 *to* 3 *Years.*	3 *to* 6 *Years.*	6 *to* 9 *Years.*	9 *to* 13 *Years.*	*Total.*
Lesions causing death	Human	55	29	19	19	15	6	143
	Bovine	1	2	2	1	—	—	6 (4 per cent.)
Lesions not causing death	Human	—	1	1	2	1	—	5

Percentage in brackets.

from the stomach washings of children with clinical and radiological evidence of pulmonary tuberculosis. The incidence of bovine strains (3·9 per cent.) which we have obtained in these cases corresponds to that (3·8 per cent.) found by Griffith and Munro (1932) for strains isolated from adult cases of phthisis in Scotland. No family history of tuberculosis was obtained in the bovine-infected cases, while this was positive in 29 per cent. of the children with human infections.

Prognosis and Diagnosis.

We have found that in children under three years of age primary thoracic tuberculosis was nearly always fatal. Accordingly, the prognosis is grave in this type of case.

We are not competent to speak on the clinical diagnosis of such cases, though from a comparison of the findings before death and those obtained at autopsy in the above series of 2,500 cases it appeared to us that for an accurate diagnosis it is essential to correlate the clinical findings, any history of exposure to tuberculous infection, the radiological picture of the chest, and the results of a carefully performed tuberculin test. From our experience of post mortem examinations in children we have been impressed by the fact that many of the hilum and lung shadows found on X-ray examination are not necessarily tuberculous. The tracheo-bronchial glands of the child respond very rapidly to any acute or chronic inflammatory pulmonary lesion, and, rapidly enlarging on account of hyperplasia, cast shadows. On the other hand, such lesions as solitary cavities in the lung substance are very suggestive of tuberculosis, particularly if these occur in cases with

positive tuberculin tests, though such lesions as bronchiectasis, secondary abscesses in broncho-pneumonia, and the more rarely occurring congenital lung cysts must be kept in mind. In cases of bronchiectasis there is usually decided enlargement of the tracheo-bronchial glands, while in simple broncho-pneumonia the same holds good even for some time after the lung lesion has cleared up. Any lesion which is undergoing calcification—a very rare condition in our experience—on the other hand, casts very definite shadows, and thus is highly suggestive of tuberculous disease, although even here it is possible for old inflammatory lesions in which necrosis has taken place to calcify. When there is generalisation of the disease in the lungs, the " snow-storm " lung appearance found radiologically is typical, and is caused by the numerous subacute miliary tubercles and small nodules of tuberculous broncho-pneumonia. But even here we have seen confusion arise in the rarely occurring cases of bronchiectasis in which the radiological picture is closely similar.

The finding of tubercle bacilli in the sputum or stomach washings will, of course, clinch the diagnosis in favour of tuberculosis, but as we have already stated, in our experience only a few cases gave positive results, and half of these died within a very short time. In this connection our results are also at variance with those of Continental observers, who have been able to isolate tubercle bacilli from the stomach washings of a large percentage of cases of suspected pulmonary tuberculosis. Too often indeed is tuberculous meningitis or generalised miliary tuberculosis the first sign of tuberculous pulmonary disease in the young child, and to avoid this grave secondary sequel a wider application of the tuberculin test in young children is desirable. When this is positive, the child should be regarded as a case of incipient tuberculosis and an attempt made to locate the seat of the disease and treat it by the usual antituberculosis measures, and the exhibition of remedies rich in vitamin D in order to promote calcification in the early lesion.

Primary Abdominal Tuberculosis.

One great difference between tuberculosis on the Continent and in America, as compared with Great Britain, is the very much greater frequency of abdominal tuberculosis in the last-named. Primary tuberculous lesions were found in the abdomen of 82 (3·9 per cent.) of the 2,126 cases under three, and in 41 (11·0 per cent.) of 374 over this age. Thus, as in primary thoracic tuberculosis, the relative incidence of primary abdominal lesions to the total number of necropsies was higher in children over three than in those under. Of the lesions which had not caused death, 8 (9·8 per cent.) occurred in the first three years of life, and 24 (58·5 per cent.) in children

over this age (Table VIII.). In 5 of these 32 cases there were, in addition, primary thoracic lesions which had caused death. Thus, as compared with thoracic, tuberculous abdominal lesions were much less fatal, especially in children over three years. As regards the types of primary lesions in this series, it was found that intestinal ulceration with caseation of the mesenteric glands occurred in 22 instances. All these cases were fatal and the majority occurred in children under three (Table VIII.). In the remaining 101 cases, though the mesenteric glands were tuberculous, no naked-eye evidence of intestinal ulceration was noted. The glands chiefly involved were the mesenteric, though in 3 cases the position of the glandular lesions was unusual and worthy of mention. In these subjects the mesenteric glands appeared healthy, but those around the head of the pancreas were enlarged and caseous. In 77 instances the glands showed no evidence of

TABLE VIII.

TYPES OF LESION AND RELATION TO CAUSE OF DEATH IN CASES OF PRIMARY ABDOMINAL TUBERCULOSIS.

Type of Lesion in—		*Result of Lesion.*	*0 to 1 Year.*	*1 to 2 Years.*	*2 to 3 Years.*	*Total 0 to 3 Years.*	*3 to 13 Years.*
Intestine.	*Glands.*						
22 cases with ulcers ..	No healing	Death	12	3	4	19 (23·2)	3 (7·3)
101 with no ulcers	Healing	Death	—	2	1	3 } 6 (7·3)	3 } 18 (43·9)
		Not cause of death	1	—	2[2]	3 }	15[2] }
	No healing	Death	17	24	11	52	11
		Not cause of death	—	3	2[1]	5	9
Total	.. ..	.. ..	30	32	20	82	41

Percentages in brackets. Small figures indicate cases in which there was also a primary thoracic lesion.

healing, and the majority of these cases (57) were under three years. In 24 subjects there was evidence of arrest, either fibrosis, calcification, or both, and most of these cases (18) were over three years of age. Thus, as in primary thoracic tuberculosis, evidence of arrest of the abdominal lesions increased with the age of the child. On considering the other tuberculous lesions present in these abdominal cases which died as a result of the disease, we found that in 12 instances there had been extension of the glandular lesion through the lymphatics of the diaphragm into the thoracic glands. This was more frequent in children under three years of age. Generalisation—namely, tuberculous meningitis, tuberculomata, generalised miliary tuberculosis, or various combinations of these—was responsible for death in

the majority of the children dying as a result of the primary abdominal infection, 61 (74·4 per cent.) of 82 cases being under three, and 12 (29·3 per cent.) of 41 being over three (Table IX.). The disease, when confined to the abdomen, caused death either on account of tuberculous peritonitis (mostly) or extensive intestinal ulceration in 11 (13·4 per cent.) of those under three years, and in 5 (12·2 per cent.) of those over this age. The percentage of deaths due to generalisation of the disease was thus very much greater in children under three years than in those over this age, while that due to disease localised to the abdomen was practically the same in children under and over three years. As compared with primary thoracic

TABLE IX.

CAUSE OF DEATH IN CASES OF PRIMARY ABDOMINAL TUBERCULOSIS.

Lesions.	0 *to* 1 *Year.*	1 *to* 2 *Years.*	2 *to* 3 *Years.*	*Total* 0 *to* 3 *Years.*	3 *to* 13 *Years.*
Cerebral, generalised miliary tubercle, or both	22	25	14	61 (74·4)	12 (29·3)
Bone	2	—	—	2 (2·4)	—
Peritonitis	5	4	2	11 (13·4)	4
Intestinal ulceration	—	—	—	—	1 (12·2)
Not cause of death	1	3	4	8 (9·8)	24 (58·5)
Total	30	32	20	82	41

Percentages in brackets.

tuberculosis, more deaths resulted from local abdominal tuberculosis (16 of 123 cases, or 13·0 per cent.) than from tuberculosis localised to the lungs and thoracic glands (8 of 239 cases, or 3·3 per cent.). In only 13 per cent. of the abdominal cases as compared with 29 per cent. of the thoracic was a positive history obtained of tuberculous disease in another member of the child's family. The reason for this becomes apparent when the type of bacilli isolated from the lesions is considered.

Types of Bacilli found in the Primary Abdominal Cases.

In 58 cases dying as a result of abdominal tuberculosis, the bacilli were isolated and typed direct from the abdominal lesions in 51 cases, and in 7 from the cerebro-spinal fluid of patients dying of tuberculous meningitis in whom primary abdominal lesions were found at autopsy. Of these strains, 11 were human and 47 (81·0 per cent.) bovine (Table X.). In children under three the bovine percentage was 80·9, and in those over three 81·8. In 15 children not dying as a result of the abdominal lesions,

the infecting organisms were also isolated and typed, and consisted of 2 human and 13 (86·7 per cent.) bovine strains (Table X.). Thus, as compared with thoracic tuberculosis, the bovine percentage in abdominal tuberculosis was very much higher. This type of infection, which is particu-

TABLE X.

TYPES OF BACILLI ISOLATED FROM CASES OF PRIMARY ABDOMINAL TUBERCULOSIS.

		0 *to* 1 *Year.*	1 *to* 2 *Years.*	2 *to* 3 *Years.*	3 *to* 6 *Years.*	6 *to* 9 *Years.*	9 *to* 13 *Years.*	*Total.*
Lesions causing death	Human	3	5	1	2	—	—	11
	Bovine	15	16	7	5	1	3	47 (81·0)
Lesions not causing death	Human	—	—	—	1	1	—	2
	Bovine	1	2	1	5	4	—	13 (86·7)

Percentages in brackets.

larly prevalent in the early years of life, is derived from milk, but why British children should suffer so extensively from infection with the bovine bacillus as compared with Continental is a problem very difficult to explain and worthy of discussion. It may, like the greater death-rate from thoracic tuberculosis, be due to dietetic, climatic, and racial factors.

Prognosis and Diagnosis.

As we have observed from our autopsy experience that abdominal tuberculosis is less fatal than thoracic, it follows, therefore, that in the former the prognosis is better. The diagnosis of early abdominal tuberculosis—*e.g.*, glandular lesions—is difficult, though in children the administration of a general anæsthetic may facilitate the palpation of enlarged abdominal glands. Care, however, must be exercised to ensure that the masses felt are not due to fæcal accumulations or neoplasms. As regards the latter, it must be remembered that, as we have pointed out (1934), the abdomen is the most frequent site of neoplastic disease in the child. Radiological demonstration of enlarged caseous glands in the abdomen is more difficult than in the thorax, though in the former calcium deposit is more frequent and may cast dense shadows. Not infrequently, however, as in thoracic tuberculosis, the onset of tuberculous meningitis is the first sign of the abdominal mischief. At other times enlarged caseating glands are discovered by the surgeon during laparotomy for some acute abdominal condition. The cardinal features of tuberculous peritonitis are well known and need not be discussed here.

Cervical Tuberculosis.

Only 8 cases of primary tuberculous cervical adenitis were found in this autopsy series, a fact testifying to the benignity of this relatively common type of tuberculosis in children. Of these cases, 4 were under three years and 4 over this age; 2 (1 under three and 1 over) did not die as a result of the tuberculous disease. In the remaining 6, generalisation of the infection —tuberculous meningitis, tuberculomata, or generalised miliary tuberculosis—caused death. From these cases of tuberculous cervical adenitis, 5 strains (3 human and 2 bovine) were obtained from children who died as a result of the glandular lesion, while 1 bovine strain was isolated from the glands of a child under one year, who did not die from tuberculosis.

Primary Site of Infection Unknown.

In this group were 7 cases, and these have already been fully reported by us (1932). They comprised 2 cases of bone tuberculosis (lumbar and cervical vertebræ respectively), 2 with tuberculous meningitis in which no other focus of infection could be found, and 3 cases where only a limited examination was permitted. Of these 7 cases, tuberculous meningitis had accounted for death in 4, tuberculosis of the cervical vertebræ with compression of the spinal cord in 1. From 3 of the meningitis cases human strains were isolated.

Summary.

The present investigation is based on a survey of 2,500 consecutive autopsies on children, in whom a thorough search for evidence of tuberculous disease was made. The relative incidence of tuberculous lesions to the number of autopsies was less in children in the first three years of life than in children between three and thirteen, though it was in the third year that the peak incidence occurred. Girls at all ages were more often affected than boys. The yearly incidence of positive tuberculin tests in a large number of children who came to autopsy practically corresponded with the incidence of tuberculous lesions found. The tuberculous lesions were the cause of death in 240 (95·2 per cent.) of 252 subjects under three and in 93 (74·4 per cent.) of 125 over this age. In 5 cases there were double primary sites of infection, 1 in thorax (responsible for death) and 1 in the abdomen.

The primary site of infection was most commonly thoracic, and then, in order of frequency, abdominal, cervical, or unknown. Primary thoracic tuberculosis occurred in 63·4 per cent. of the tuberculous cases, and relative

to the number of autopsies had a higher incidence in subjects over three years, but was more fatal under this age. Only 0·6 per cent. of the cases under three and 9·0 per cent. over this age with primary thoracic tuberculous lesions did not die as a result of the tuberculous infection. Support for this finding is derived from (1) observations on adult autopsies, (2) tuberculin tests in children, (3) clinical findings and results of stomach washings. Primary lung lesions of more acute progressive type were found in children under three years than in those over, while evidence of arrested lesions was commoner in the older children. The primary lung lesion was always associated with tuberculous tracheo-bronchial glands and was very rarely the sole tuberculous lesion in the lungs. Most of the children with primary intrathoracic tuberculosis died of generalised infection, and this was slightly commoner in children under three than in those over.

In 149 children who died, the infection was due to human strains in 143 and bovine in 6 (4 per cent.). There was a slightly higher percentage of bovine strains in children in the first three years of life than in those between three and thirteen. From children not dying as a result of the tuberculous lesion, 5 human strains were isolated.

The prognosis is grave in cases of primary intrathoracic tuberculosis, and for a diagnosis it is necessary to correlate the clinical and radiological findings, the family history, result of tuberculin tests, and examination of sputum or stomach washings. A wider general application of the tuberculin test in young children is recommended in order to detect any positive reactors.

In 123 (32·4 per cent.) investigated, the primary site of the tuberculous infection was abdominal. The relative incidence of primary abdominal lesions to the total necropsies was greater in children over than in those under three years, but more deaths resulted from the abdominal infection in children under three. Intestinal ulceration was present in 22 abdominal cases; such lesions were always fatal and were commoner under three years. In 101 instances there was no tuberculous bowel lesion, but the mesenteric glands were caseous. Evidence of healing in these glands was commoner in children over three years. In 74·4 per cent. of children under three and 29·3 per cent. of those over this age generalisation of the infection caused death. Tuberculous disease localised to the abdomen caused more deaths than similar disease confined to the lungs.

Of the bacilli isolated from the abdominal cases in which death resulted from tuberculosis, 11 were human strains and 47 (81·0 per cent.) bovine. From children not dying as a result of the abdominal disease, 2 human and 13 bovine (86·7 per cent.) strains were obtained.

The prognosis in primary abdominal tuberculosis is better than that in

primary thoracic tuberculosis, but the diagnosis of the abdominal disease in the early stages is often attended with much difficulty.

Primary tuberculosis of the cervical glands was not often found in this autopsy series, occurring in only 8 (2·1 per cent.) cases, of which 2 did not die as a result of the lesions, the remainder dying of generalisation of the disease. From this group 3 human and 3 bovine strains were isolated. In 7 (2·1 per cent.) subjects no primary source was found, and from these 3 human strains were obtained.

TABLE XI.

SUMMARY AND AGE DISTRIBUTION OF 236 STRAINS ISOLATED FROM CHILDREN IN THIS SERIES.

Type.	*0 to 1 Year.*	*1 to 2 Years.*	*2 to 3 Years.*	*3 to 6 Years.*	*6 to 13 Years.*	*Total.*
Human	59	36	22	27	23	167
Bovine	19	20	10	12	8	69
Bovine per cent.	24·4	35·7	31·3	30·8	25·8	29·2

Bovine per cent. 0 to 3 years = 29·5.
Bovine per cent. 3 to 13 years = 28·6.

Table XI. summarises the various strains isolated from children in this series. It will be noted that the bovine percentage rises to its peak (35·7 per cent.) in the second year of life, and thereafter gradually falls to 25·8 per cent. in children between six and thirteen years. The percentage of bovine infections is practically the same in children under as in those over three years of age.

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REFERENCES

J. W. S. BLACKLOCK: *Med. Res. Counc. Spec. Rep. Series*, No. 172, 1932.
J. W. S. BLACKLOCK: *Trans. Roy. Med. Chir. Soc.*, Glasgow, 1934, **28**, 127.
A. S. GRIFFITH AND W. T. MUNRO: *J. Path. Bact.*, 1932, **35**, 271.
P. D'ARCY HART: *Med. Res. Counc. Spec. Rep. Series*, No. 164, 1932.
J. C. HENDRIE: A Study of Tuberculous Infection in Infancy and Childhood. *Thesis for M.D. Degree, Glasgow*, 1933. Unpublished; in the archives of the University library.

NATURAL CURE OR COLLAPSE THERAPY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

By M. JAQUEROD,
M.D.,
Medical Director of Society of Climatology, Leysin, Switzerland

Are we still right in treating pulmonary tuberculosis by rest alone, without at once inducing a pneumothorax or employing some other form of collapse therapy? About twenty years ago treatment by collapse, then limited to such methods as artificial pneumothorax and thoracoplasty, were applied only to advanced cases which did not respond to simple medical means. For others with a more favourable prognosis, rest, fresh air, and the treatment of symptoms were advocated. Under these conditions the results of collapse, with a few striking exceptions, were often mediocre and deceptive.

About the same time the advance made in radiology made it possible not only to follow out the stages of a cure from the onset of disease to the disappearance of all signs and symptoms, but it also showed that cures effected after a long period of rest were much more complete than one formerly supposed. Besides the well-known cures effected by the formation of fibrosis it was found that extensive areas of infiltration dried up without leaving any scars, and even cavities of moderate size closed in a comparatively short time.

The natural cure for cases of recent and localised disease thus gave much better results than those treated by collapse, and a large number of old cases whose disease had been arrested for many years were a living proof of the solidity of a cure effected by rest alone.

Little by little, however, the contra-indications to artificial pneumothorax were set aside and it began to be induced for the early and slight case, and in consequence the results of this new method of treatment improved to such an extent that today a large number of specialists confronted with a case of pulmonary tuberculosis, whatever the type of lesion or its clinical significance may be, consider it the duty of a doctor to induce a pneumothorax at once, or, if this is impossible, to try one or other of the methods of collapse, such as phrenicectomy, extra-pleural plombage, or thoracoplasty.

It is obvious that in this way a cure occurred in a large number of patients who would have been equally cured by rest alone. What advantage has artificial pneumothorax over more simple methods in such cases? In other words, are the cures effected by pneumothorax more rapid, easier and more lasting than those effected by placing the patient under the best conditions to obtain a natural and spontaneous cure?

In order to answer this question it is necessary to make a fundamental distinction between the case of recent activity and the old chronic one. The process of healing differs completely in these two types. In the first the lesion may heal, almost if not altogether, by resolution, which restores the lungs to their original condition. If, however, there are recent cavities caused by the destruction of lung tissue, then in addition to resolution there is another process consisting of the collapse of the diseased areas, with the consequent drawing together of the walls of the cavities and the fibrosing of the lesions. This is exactly what happens when the disease is treated by pneumothorax, but it is brought about by a diametrically opposite method.

With a pneumothorax the whole of the lung is collapsed in order to obtain contraction of the diseased part. Under certain favourable conditions, however, it is possible to obtain a selective collapse—that is to say, collapse of the diseased lobe only—but the necessity of giving regular refills impairs the function of the healthy lung, so that a cure by artificial pneumothorax is nearly always bought at the price of a considerable reduction in the original volume of the lungs.

On the contrary, the natural cure leads to compensatory hypertrophy of the healthy lung, which begins to compress and overlap the excavated and damaged areas and to occupy their place in the thorax. Recovery of function is thus better and more complete after a natural cure than after one obtained by pneumothorax.

Let us now consider whether in recent cases, equally suitable for either method, a cure by pneumothorax is obtained more quickly and easily than by simple natural methods.

A natural cure necessitates absolute rest for the patient under favourable conditions as regards fresh air and food for one or two years at least. Thus it may well seem that the first advantage of pneumothorax is that it will obviate this long period of inaction, and perhaps the time will come when consumption hospitals and sanatoriums will be vacated and replaced by dispensaries where patients may go for refills two or three times a month and return immediately to their various occupations. Unfortunately, things have not proved quite so simple as one hoped. Complications often occur when a patient returns too soon to work, affect seriously the result of treatment, and often necessitate a return to prolonged rest. Today

the most enthusiastic advocates of collapse therapy are loud in their protestations that surgical procedures are but adjuncts and in no way dispense with the standard method of cure by rest, hygiene, and diet.

During the first two years the consumptive treated by pneumothorax remains, so to speak, a sanatorium patient. During the third year caution is still necessary, but after this complications are rare and the patient usually returns gradually to a normal life. Refills, however, must be continued for three or four years, and some authorities take the extreme view that in the absence of contra-indications the lung should be kept permanently collapsed. Thus it is quite obvious that the idea of gaining time and obtaining a quick cure has nothing to do with the induction of a pneumothorax, and from that point of view one is perfectly justified in suggesting that a patient should try simple medical means when there is a reasonable chance of obtaining a spontaneous cure in this way.

It is a matter of first importance to decide whether or not a spontaneous cure is possible, and this is one of the most difficult questions to answer. In practice it is impossible to decide after a single examination, and a period of observation is necessary before the physician can pass judgment. Apart from medical considerations, the problem depends largely on the ability of the patient to devote himself to strict treatment by rest, for it must be remembered that there is a definite risk that the patient will not keep strictly to rest, in which case he is safer with a collapsed lung. Consequently pneumothorax should be induced without hesitation when for one reason or another the patient cannot immediately and thoroughly devote himself to undergoing a natural cure. The same applies when the slightest loss of time would be disastrous for the patient, owing to such grave symptoms as high temperature, profuse hæmoptysis, or rapid loss of weight. These are emergencies which call for the immediate induction of a pneumothorax. There is no time to lose and no choice of treatment, because intervention offers the only hope of stopping the spread of the disease.

If, however, such reasons do not exist, there is no immediate hurry to induce a pneumothorax, and the patient should be kept at rest, and the signs and symptoms watched carefully day by day and week by week in order to follow the development of the disease, the temperature, weight, sputum, and X-ray findings, and if all goes well there is no reason to interfere. Any healing which takes place naturally is a definite step forward towards complete cure, which will eventually become established if the patient has sufficient patience and perseverance.

On the contrary, if after some weeks of observation there is no improvement, or only a small amount of fibrosis has formed and the condition appears to be stationary, then intervention is indicated.

It would be illogical not to give the patient with a recent pulmonary lesion the chance of improving with simple medical means, which in a number of cases is all that is necessary to obtain a complete cure in the minimum of time and with the minimum of organic and functional disturbance.

The case is different, however, when we consider the chronic case of several years' duration and with tubercle bacilli constantly present. Formerly, before the introduction of collapse therapy, such patients were treated by simple medical means only, or perhaps with one or other of the innumerable remedies which were constantly being pressed as cures for the disease, remedies sometimes helpful but more often useless. A large number of such patients lived to a good age and maintained a fair standard of health for a number of years in spite of positive sputum, others gradually lost ground, and in a certain number the disease became completely arrested after some years. Such cures were effected by fibrosing of the infiltrated areas, and in some cases to such an extent that the whole lung was transformed into a solid mass of fibrous tissue. Even today I still see a certain number of these patients who in their youth had spent years in some sanatorium and whose disease has remained arrested for twenty years or more.

The process of healing in these cases in no way differs from that brought about by collapse therapy, except that the latter is more certain and more thorough. One should not hesitate, therefore, nowadays to advise surgical intervention to such patients unless, of course, there are definite contra-indications, such as advanced age, complications in another part of the body, etc.

Now, since it is admitted that a pneumothorax should be induced when it is seen that the patient is not improving with simple medical treatment, it would seem at first sight that the chronic fibroid case so often met with formerly should not be in evidence today. This would be so if pneumothorax gave perfect results, but this is far from being the case, and I do not think I exaggerate when I say that the chronic fibroid case is as common today as before the introduction of collapse therapy.

Moreover, one must take into account the considerable number of cases in which pneumothorax is not possible and those in which an insufficient collapse is maintained for years. Both these types are apt to lead to some surgical procedure complementary to artificial pneumothorax, such as phrenicectomy or division of adhesions. These operations are themselves often ineffective or incomplete, and one has only to glance at statistics to see how unsatisfactory are the results, and so, sooner or later, it becomes necessary to proceed with the series of surgical methods, and the patient comes to one or other of the more drastic forms of treatment—plombage,

partial thoracoplasty, or complete thoracoplasty. Even so the results are not satisfactory, except in a very limited number of cases.

As a result of this long series of surgical interventions, the hospitals and sanatoriums are full of chronic cases which bear testimony to the fact that even now healing is often imperfect and unsatisfactory in spite of the enormous advance which has recently been made in the treatment of pulmonary tuberculosis by collapse therapy.

When confronted with a case of recent disease, and one weighs in the balance the advantages of treatment by rest alone and treatment by artificial pneumothorax, it is not right to infer that the one offers but a mediocre chance of cure, whereas the other gives the patient almost a certainty of a rapid and complete recovery. For in a considerable number of cases (and it is impossible to foretell which) treatment by pneumothorax will only mean a series of painful procedures continued over a period of years.

In conclusion I would answer the question put at the beginning of this paper by submitting that the custom which would enforce collapse therapy at once for all cases of pulmonary tuberculosis is not justified, for there are cases which reap the greatest possible benefit by being given the opportunity of obtaining a lasting cure by simple and natural means.

THE TREATMENT OF CAVITIES IN PULMONARY TUBERCULOSIS

By H. MORRISTON DAVIES,

M.D., M.CH.(CANTAB.), F.R.C.S.(ENG.),

Medical Superintendent, Vale of Clwyd Sanatorium; Consulting Surgeon to University College Hospital, City of London Hospital for Diseases of the Heart and Lungs, King Edward VII. Welsh National Memorial Association, Lancashire County Sanatoriums, Cheshire Joint Sanatorium.

Cavities are generally considered to be evidence that the body is able to offer resistance to the invasion of the tubercle bacilli. This is undoubtedly true, but puts an optimistic interpretation on the facts. If caseation takes place and there is excavation of the lung substance, following which there is a steady, progressive deposit of fibrous tissue, demarcating and enclosing the lung cavity, then we can rightly assert that the body is putting up a resistance against the invading organisms. This, however, is the limit of possible favourable interpretations of cavities. We certainly cannot say that the cavity will become progressively obliterated or that the lung disease will heal. In fact, the very presence of the cavity with its fibrous tissue around is evidence of a state of affairs which, while indicating a temporary localisation of the disease, threatens sooner or later a spread and ultimately the death of the patient.

There are, however, cavities which do not give us even the element of consolation of temporary resistance and localisation. They are the acute cavities: spaces without defined border or shape, and which appear on a radiogram as irregular areas of greater translucency without any defining contour—a space recognised by contrast of shadows only. Sometimes the contrast is further emphasised when the acute destruction has taken place within a zone of consolidated lung tissue. In these cases, except for the fact that the granulomata have caseated, there is no evidence of a local response or of the formation of a protective barrier.

A less acute variety is seen when there are a number of small cavities all in close proximity to each other, but not becoming actually confluent into one space. They give to the lung a " moth-eaten " appearance. These multiple cavities may eventually coalesce and may even, if the patient's resistance become sufficiently good, form a typical chronic cavity with defined walls, but possibly still showing septa traversing the space.

While the formation of a cavity and of a surrounding zone of fibrosis can be regarded as indicating a power of resistance, yet concomitant changes may show how weak and inadequate that resistance is. These changes, when present, are seen on the radiogram as shadows of varying density and thickness round the periphery of the cavity, but more pronounced round the lower than the upper border. These shadows represent zones of pneumonic consolidation, and are evidence of the intensity of the infection, tuberculous or possibly pyogenic, within the cavity and the cavity walls. They are probably not due to a spread of the tuberculous disease through the walls of the cavity, as the consolidation may clear up without leaving an appreciable scar. Such shadows are, however, of very great importance as regards both immediate prognosis and treatment.

When a cavity develops those characteristics which denote resistance to the invasion and chronicity, when, that is to say, the cavity becomes round or oval with a defined outline due to a fibrous wall, certain further considerations have to be taken into account. The ultimate fate of the cavity will be influenced by its size, its position in the lung, and the efficiency of the draining bronchus. The cavity has by this time reached the stage at which healing without artificial aid is impossible. The resistance of the body has done most of what it is capable: it has sloughed and has caused the diseased lung tissue to be thrown out, and it has built up a barrier as a protection against the further spread of the disease from that area. Once it has formed this barrier, however, it has created a mechanical obstacle to its endeavour to secure healing. The rigid wall cavity may become dry, quiescent, and apparently sterile, but for such time and only until reinfection, for whatever reason, resumes its malignant course. More often it never even temporarily reaches this stage of arrest, but maintaining its infected state it is responsible for recurrent pyrexia, for hæmorrhages, and for bronchogenic or hæmotogenous infection of other lung fields.

A cavity wall has usually a well-defined inner layer of circular fibres which, contracting, would convert the cavity into a sphere were it not for outside forces which help to mould the shape. The outer wall is not so well defined, fibrous tissue radiating out into the lung and to the pleura and to the interlobar septum when in proximity to these.

When the wall of a cavity comes into close proximity to the visceral pleura it will, sooner or later, produce inflammatory changes which result in fusion of the pleural membranes. Similarly, when it approximates the interlobar fissure it causes thickening of the septum and fusion of the adjoining lobes. These changes are of serious consequences because they interfere with healing and with treatment.

The healing of cavities is obtained by the contraction and approximation

of their walls. These changes are dependent on two factors. One is the freedom from attachments which fix the walls either to the chest wall through the fused membranes, or to the thickened interlobar fissure. The other factor is the ability of the surrounding lung tissue to compensate for the shrinkage of the cavity. It follows from this, therefore, that the more centrally-placed cavity is better situated for healing than one which is near the periphery of the lobe; and that a cavity in the apex of the lung is in the most adverse position, because, owing to the narrowness of the apex, a much greater proportion of the circumference of the cavity approximates the lung periphery.

When a cavity approaches the surface of the lung, it is not long before the inflammation produces interpleural adhesions. It is quite a common occurrence to endeavour to collapse the lung by artificial pneumothorax, and to find that the visceral pleura can be separated from the parietal membranes except over the cavity, which is usually the region it is most essential should collapse fully. Adhesions may be capable of stretching, but even while doing so they are exerting a strong traction force on the cavity wall, and therefore during the collapse of a lung by artificial pneumothorax it is no unusual thing to see the cavity suspended by adhesions and increasing in size, while the rest of the lung is reduced in volume.

A similar increase in size of the cavity may be seen when the upper lobe is universally adherent and there has been fusion of the interlobar septum and fixation of its peripheral margin to the chest wall. At first, by exerting even a very mild pressure force during artificial pneumothorax treatment, collapse of the lower lobe, or lower and middle lobes, is complete and the interlobar fissure is arched upwards towards the apex constricting the cavity. Later, as the interlobar membranes thicken still more and the fibrous tissue, contracting, takes the shortest course between hilum and periphery, it becomes a straight line between these points and will cause traction on the lower wall of the cavity and an increase in its size.

An appreciation of these facts carries with it the corollary that the nearer a developing cavity is to the surface of the lung, and particularly to the apex, or to the interlobar septum, the earlier must active treatment in the form of artificial pneumothorax be begun. Delay is to risk failure, owing to fusion of the pleural membranes. It is only when the cavity is centrally placed that one may be justified in delaying in the hope that obliteration and healing will take place by unaided sanatorium régime. But even in these cases there is risk in delay. A cavity is probably never an isolated lesion in the lung. There will be other foci more superficial, perifocal inflammation which can just as readily produce adhesions as inflammation around a superficial cavity.

It is not possible to say at what stage in the course of the disease adhesions will occur which will interfere with lung collapse therapy. Artificial pneumothorax will be found impracticable in one case with a tuberculous lung lesion of comparatively recent origin: it may be found possible, on the other hand, in an old fibrotic case of many years' duration.

When, therefore, considering whether delay in starting collapse therapy for the treatment of a centrally-placed cavity is justified or not, many points may have to be taken into consideration. The first is the cavity. If it is over 3 cm. in diameter, if there are fibrous tissue changes in the walls, if there is surrounding consolidation, if it is giving rise to hæmorrhages or is not draining properly, a pneumothorax should be attempted at once. Then there is the presence of associated disease in the same lung. If this is at all extensive, if there are active foci near the surface, or the radiogram gives evidence of caseating processes, collapse therapy is indicated.

The influence on one's decision of disease in the contralateral lung is more difficult of definition. It may be that there is a suggestion of beginning cavitation in this lung also, which may in a month or so call more urgently for collapse therapy; or there may be a moderately acute exudative invasion. In such cases the alternative between a bilateral artificial pneumothorax or an immediate pneumothorax on this side in the hope that the central cavity in the other lung can respond to bed rest, the contralateral collapse possibly supplemented by a phrenicectomy must be considered.

The extrapulmonary complications are also important deciding factors. A dry pleurisy or an effusion on the side of the cavity would indicate the need for artificial pneumothorax. Tuberculous invasion of the larynx is a most definite indication, whilst mild symptoms suggesting intestinal tuberculosis would weigh in favour of an immediate therapeutic lung collapse.

The position of the communicating bronchus in relation to the cavity is of considerable importance. If the bronchus is of adequate size and opens into the most dependent part of the cavity, drainage is free and there is the minimum of toxæmia from secondary infection. The cavity may none the less be responsible for pyrexia if there are progressive tuberculous changes in its walls. When the opening of the bronchus is at too high a level there is retention of secretions, absorption of toxins and pyrexia. Postural drainage may succeed in emptying the cavity for the time being, but the constant re-accumulation of secretions in the cavity must militate against healing.

It occasionally happens that the lumen of the draining bronchus becomes temporarily occluded by congestion of the mucosa and further escape of the secretions is impossible. The temperature rises and there is increased toxæmia. After a few days communication is re-established, there is an increase in sputum and a subsidence of the symptoms.

A knowledge of these facts is necessary before one can discuss treatment. There are three outstanding points. The one is that a cavity which has become chronic will rarely heal unless some surgical measure is undertaken to overcome the mechanical difficulties. The second is that whilst a cavity may heal spontaneously before it has developed a surrounding fibrous wall, unless this cavity is situated well within the lung substance and at a distance from the visceral pleura and the interlobar fissure, complications due to the inflammation reaching the pleural membranes will arise, and the simpler forms of surgical intervention will no longer be available. A third most important feature influencing treatment and the success thereof is the size of the cavity.

Bed rest is essential in all cases of pulmonary tuberculosis in which there is active disease. The greater the activity and the pyrexia the more absolute must the bed rest be. By bed rest alone many cases of tuberculosis can be healed, but for many others it will fail to arrest the disease. It is therefore frequently necessary to supplement bed rest by surgical measures, either because rest alone is inadequate, or because healing can be expedited or the future can be rendered more secure.

Even when cavities are present, the obliteration of the cavity and healing may be secured by bed rest alone. This applies more especially to the acute early cavities with ill-defined walls. As the surrounding cavity walls become more sharply demarcated by surrounding fibrous tissue changes and the cavity develops a circular or oval outline, and as the cavities increase in size to over 2 cm. in diameter and more, so does the possibility that bed rest alone will suffice to arrest progress and produce obliteration become progressively less. Fales and Beaudet[1] found in their more recent series of 90 cases, of which 92·7 per cent. were between thirty-one and forty-five years of age, " that the young or exudative cavities responded most favourably to bed rest (66 per cent. healed); the moderately old or productive cavities less favourably (43 per cent. healed); and the old or fibrotic cavities unfavourably (only 17 per cent. healed)." These figures are exceptionally good, and must indicate a very complete bed rest continued over a long period. Such a result could, of course, not be obtained with a younger age-group.

The difficulty is to decide when and for how long bed rest alone may be profitably tried. With a large cavity surrounded by a thick fibrous wall it is a waste of time—provided always surgical measures are not contra-indicated by disease in other lung-fields—unless a period of intensive rest is needed to build up the general health of the patient prior to more active treatment. The same is true of the larger cavities of the productive type surrounded by a thin zone of fibrous tissue, and also of all cavities in young

adolescents. The small cavities of the productive type and the exudative cavities without well-defined walls in patients over thirty are the ones most likely to respond to bed rest. But with these also there is a difficulty: the longer bed rest is continued, the greater is the danger that adhesions will form over the surface of the cavity, and that when, therefore, it is considered necessary to collapse the lung by artificial pneumothorax, separation of the membranes is no longer possible.

In every case of pulmonary tuberculosis individual factors must always influence one in one's decisions: factors not only of age, of build, of general extent of the disease, of resistance, of associated changes such as bronchitis, but also of the mentality of the patient; yet certain general indications can be given. The younger the patient and the nearer the cavity is to the surface, the sooner must the artificial pneumothorax be started. When the patient is over thirty, when the cavity is centrally placed and when it has little or no demarcating outline, and when the disease is found to be responding to bed rest, it is safe to wait and watch for a month or two, and longer if the cavity is decreasing in size. When there is an exudative cavity of over 3 cm. in its biggest diameter, or a productive cavity of over 2 cm., valuable time and opportunity are being lost by any delay in attempting an artificial pneumothorax. With a large, thick, rigid wall cavity, especially if in the apex, it is very problematical if it is worth while even attempting an artificial pneumothorax, if the case is one for which an apical thoracoplasty is not contra-indicated.

Further difficulties in decision have to be faced when adhesions are found to interfere with the satisfactory collapse of the cavity. In such cases, as has already been said, the probability is that the suspended cavity will increase in size. Very occasionally the adhesion will stretch; but it is dangerous to attempt to do this by raising the intrapleural pressure to a positive one, especially if the adhesion is attached directly to the wall of a moderately thin-wall cavity. This wall is probably the weakest point: it may, therefore, give way and cause a spontaneous pneumothorax, a tuberculisation of the pleural cavity and a tuberculous empyema, or a mixed infection empyema. In some cases the adhesions are not too extensive and have yielded sufficiently to allow of them being divided by coagulation and cautery with thoracoscopy.* When this is not possible, the advisability of abandoning the pneumothorax and collapsing the cavity by thoracoplasty will have to be considered. Before, however, this is done the effect of phrenicectomy must be tried.

* O'Brien[2] advocates access to the adhesions by direct vision, crushing the adhesion and dividing by electro-coagulation or between ligature. He claims closure of the cavity in 11 out of 16 cases.

The combination of hemidiaphragmatic paralysis with artificial pneumothorax has proved most beneficial in all classes of cases, including those with suspended cavities. Nehil and Alexander[3] obtained complete closure of the cavities in 59 per cent. of 116 patients for whom the phrenic operation was added to an unsatisfactory pneumothorax because of the continuing presence of cavernous lesions.

Phrenicectomy is indicated for those cases on which an artificial pneumothorax has been tried, but has failed to produce any material collapse. The success or otherwise of the hemidiaphragmatic paralysis will depend on many factors. The paralysis of the muscle is in itself of value, as it abolishes the inspiratory drag on the fibrous wall of the cavity. The extent of the rise of the dome will determine the reduction in the hemithorax. But the distribution of this reduction available for collapsing the lung is in turn dependent on the character of the changes in the lung and on the mobility of the mediastinum. If the mediastinum has been drawn over to the affected side by the disease, restoration to a more normal position will in part counteract the effect of the rise of the dome; so, too, at a later period may a compensatory increased inspiratory action.

Thickening and fixation of the basal pleura and of the interlobar septa will interfere with the extent to which the upper lobe can benefit; as also will consolidation or caseation changes in the lower part of the lung, or dense fibrosis of the upper lobe. The type of upper lobe cavity most likely to benefit is when, the mediastinum being fixed and the pleural membrane changes being minimal, there is a moderate degree of fibrosis in the upper part of the lung round a comparatively thin-walled cavity of not more than 3 or 4 cm. in diameter.

The following figures given by Nehil and Alexander show the effect of phrenicectomy on cavities in 179 patients, according to the size of the cavities:

Size of Cavity.	*Cavities.*	*Closed.*	*Smaller.*
	No.	*Per Cent.*	*Per Cent.*
Moth-eaten	11	27	9
0-2 cm.	53	65	2
2-4 cm.	63	36	27
4-6 cm.	32	22	31
6-8 cm.	9	12	—
8-10 cm.	5	—	20
10 cm. plus	6	—	—
Total	179	38	16

They found, moreover, that while 40 per cent. of cavities in the upper lobe closed after phrenicectomy, 34 per cent. only of those in the middle lobe and 29 per cent. of lower lobe closed. Slavin,[4] however, states that

" cavities situated in the lower lobe show more initial compression and obliterate more rapidly than upper-lobe cavities."

In every case there must be a large element of uncertainty as to the result of phrenicectomy, but the operation is so comparatively trifling a one that it is justifiable in the hope that some benefit, if not complete closure of the cavity, will result. The prospect of a successful issue in the larger cavities is, however, so doubtful that hemidiaphragmatic paralysis should only be done as a preliminary to an apical thoracoplasty or an apicolysis with paraffin filling. Its value in these cases lies in the removal of the inspiratory drag, in the steadying effect on the lower part of the lung, and the possibility that this may help to counteract the downward drop of an apical cavity after a localised thoracoplasty; the raised dome may, moreover, reduce the size of the cavity and so modify the extent of the subsequent operation.

The operation of thoracoplasty is reserved for the chronic cavities which have failed to respond to simpler methods of treatment. The type of thoracoplasty, whether apical with extensive resection of the ribs, or an eight rib resection, or a ten or eleven rib operation with the removal of moderate lengths of bone, or with an extensive first stage and less extensive second or even third stage, will depend on the size of the cavity, the thickness of its walls, and the extent of disease in the rest of the lung. When an apical thoracoplasty only is done, the tendency for the apex of the lung and for the cavity to be displaced downwards must always be borne in mind. To counteract this it is necessary that the rib resection should extend well below the cavity and that a very considerable length should be removed from the lowest rib resected. If, despite this, the cavity escapes below the collapsed chest wall, a more extended second stage resection will be needed some two to three weeks later.

A thick wall cavity may fail to collapse completely even with an extensive paravertebral thoracoplasty, and then, owing to the persistence of symptoms, still further treatment is required. Additional collapse must be produced by either an antero-lateral thoracoplasty, excising the remainder of the ribs and their costal cartilages, or by an extrapleural paraffin fill.

Extrapleural pneumolysis has its own independent value for those cases on which a thoracoplasty cannot be done. A thoracoplasty is contra-indicated in the presence of active disease or of moderately extensive fibrosis in the opposite lung. It becomes an increasingly severe risk after the age of about forty-five, depending on the type of build and general constitution of the patient. The co-existence of extrathoracic tuberculosis or of disease such as diabetes may also contra-indicate the major rib resection, but does not necessarily debar an extrapleural pneumolysis. This operation carries

with it a very small operative risk and makes but a small demand on the constitution of the patient. It is an operation, however, which is much more limited in its applicability by the site and extent of the lesion. Its greatest value is for the smaller cavities in the upper part of the upper lobe, and preferably for supraclavicular, but also for infraclavicular cavities. When the lower margin of the cavity extends below the level of the third costal cartilage, the probability of successful closure is considerably diminished.

The space produced by the extrapleural cleavage is filled with paraffin. A space which requires an amount of paraffin of more than about 200 c.c. in order to obliterate the cavity is much more liable to the risk of complications than a smaller one. Success is dependent also on the satisfactoriness of the cleavage: this may be interfered with by extrapleural adhesions which prevent adequate collapse of the cavity walls.

Access may be obtained by the anterior but preferably by the posterior route. McIndoe and Alexander[5] obtained closure of the cavity in 85·5 per cent. of 35 cases operated on by the posterior route in which satisfactory cleavage was obtained, but in 50 per cent. only of anterior route operations.

REFERENCES

1. Fales, L. H. and Beaudet, E. A.: *Amer. Rev. Tuberc.*, 1934, xxx. 225.
2. O'Brien, E. J.: *J. Thoracic Surg.*, 1932, i. 603.
3. Nehil, L. W. and Alexander, J.: *J. Thoracic Surg.*, 1933, ii. 549.
4. Slavin, P.: *Amer. Rev. Tuberc.*, 1934, xxix. 629.
5. McIndoe, R. B. and Alexander, J.: *Amer. Rev. Tuberc.*, 1934, xxix. 270.

APICOLYSIS AND PLOMBAGE IN THE TREATMENT OF TUBERCULOUS CAVITIES

By F. HÄBERLIN,
M.D.,
Medical Superintendent of the Zurcher Heilstätte, Clavadel.

With the Collaboration of BERNARD HUDSON,
M.D., M.R.C.P.,
Medical Superintendent of the Victoria, British Sanatorium, Davos-Platz.

The various therapeutic surgical methods developing from the original idea of collapsing a diseased tuberculous lung (Forlamin, 1882) have given rise to six main interventions, three of which are intrapleural operations—viz., "pneumothorax," "Jacobæus" (*e.g.*, cauterisation of adhesions), and "oleothorax"; and three extrapleural—viz., "phrenic avulsion," "thoracoplasty," and "apicolysis."

Whilst most of these operations have earned for themselves undisputed fame, this is not so in the case of the last named—apicolysis. The popularity of this operation, possibly because it combines such brilliant promise with such definite chances of failure, has waxed and waned to a remarkable degree according as one or other of these two characteristics has been in the ascendant. Apicolysis has never entirely lacked champions, but in the past this championship has only too often proved a short-lived thing when some misfortune has overtaken the operation. Hence, the operator who wishes to practise a method, the results of which are so variable, would be well advised to make serious and painstaking study of the pitfalls, in order that he may avoid them.

There is no need to dwell on its good points; the selectivity of the collapse—that is, a local collapse of the diseased parts of the lung, with conservation of all the healthy tissue—is a most important feature. There can be no question that the trend of modern phthisiology is in favour of this selective collapse. One hesitates to sacrifice the whole lung for just a small apical cavity. Furthermore, the possibility of applying the method for cavities in the apex of a lung whose collapse has been resisted by adhesions gives to apicolysis a wide range of usefulness, which should be employed to augment other methods rather than to rival them. In these cases of apical

cavities, a decision in favour of apicolysis rather than thoracoplasty was much simpler to make before the so-called "apical thoracoplasty" (Graf), or "topographic" (Leysin school), entered the field of competition. These partial plastics, which consist of complete resection of the uppermost ribs, but limited to four or five ribs, can replace apicolysis in some measure, but they rarely produce such a complete collapse. This is not merely the expression of my personal experience, but also of that master of thoracic surgery, Sauerbruch of Berlin, and of many other phthisiologists (Burrell, *Lancet*, February, 1933). Moreover, in spite of the lesser benefit following upon these partial operations, they are more dangerous, as is evidenced by their much higher post-operative mortality, whereas the absence of this mortality is a conspicuous feature in apicolysis. The shock of the operation is so insignificant that apicolysis can be safely tolerated even when, in bilateral cases, it is performed on both sides; and whilst a successfully performed bilateral plasty is rather a rarity, I myself have performed nine operations for bilateral apicolysis with only one fatality. This patient died from acute œdema of the lung, a totally unexpected accident, which, by a more careful observation of the patient's heart condition, might possibly have been avoided. Too much confidence had perhaps been induced by the uneventful post-operative course of all our previous cases. The respective risks of the two methods cannot better be demonstrated than by a comparison of the post-operative condition of the patients. After a thoracoplasty, the patient not uncommonly remains in a state of shock for several days; he feels miserable, is in great pain, and has much cough and expectoration, acts which are difficult to perform owing to the pain which they initiate. Day and night the patient must be watched by a specially trained nurse, who, to relieve the strain upon the heart, must assist the patient when coughing is necessary. The plombage patient, on the other hand, presents quite a different post-operative picture. There is no shock, little or no pain, and no difficulty with cough or expectoration (the thoracic wall not being severely injured), facts which make the incidence of aspiration pneumonia an almost unknown complication, whilst in the plastics it is the one most to be feared. The general state of the patient never becomes alarming, and no special nurse is required, the patient usually being able to take care of himself without assistance. Whoever has seen these two pictures cannot fail to appreciate the difference, and it is a fact that the merits of the latter are recognised not only by the doctor, but by the patients themselves. At my sanatorium they will accept the proposition of a plombage just as they do that of a phrenic avulsion, whereas the idea of a plasty comes as a terrible blow—they realise the danger.

What, then, are the objections that have been advanced against apico-

lysis and plombage? The most important one is certainly the fear of the foreign body. Though we can state now, secure against contradiction in the future, that an aseptically and antiseptically (for this latter see the important works of Lange of Munich concerning artificial silk tendons) implanted foreign body will be tolerated, we nevertheless cannot conceal that this remains the chief fear of every surgeon who is performing plombage operations. My personal experience of nearly 100 cases confers on me the right to state that all plombage cases can heal by first intention, and that paraffin is well tolerated. If in medical literature we find a report of about 100 cases with more than a 30 per cent. suppurative incidence (Flora Eiselsberg) we cannot but deplore a faulty technique. But the principal element of doubt lies in the more distant future. Time only will give a pertinent answer to the question of indefinite toleration of these foreign bodies. Thus Winternitz, a surgeon who very much favoured apicolysis, had to report 11·4 per cent. of tardy perforations of the paraffin into the lung. This complication, which is by no means fatal and does not always even prejudice the cure, usually necessitates the removal of the paraffin, with resection of the ribs round the plombage bed, and the covering of the lung fistula with a muscle flap. So far we have had two cases, in one of which the patient recovered without our help, after having coughed up paraffin during a few months; in the second case it occurred several weeks before the death of the patient from progressive tuberculosis, the plombage, in this instance, having failed to close the cavity. The perforation, therefore, was not the cause, but rather the result of the ultimate breakdown of tissue resistance. Nevertheless the possibility of this complication must be borne in mind. There is one important technical rule which should prevent this occurrence, or at least render it very rare. This is that one ought not tightly to pack the space obtained by apicolysis, or even attempt to fill every part of it. The small spaces left here and there will eventually fill with blood and serum, which later become organised and fibrosed, forming a kind of separation between the paraffin "sausages," and helping to fix the plombage to the thoracic wall. I am quite sure that discretion in filling the extrapleural space accounts largely for the almost constant absence of excessive exudation into the bed of a plug so inserted, which, if present, causes fever, a feeling of pressure, and enlargement of the radiological shadow. One should always remember that complete apicolysis is the main, and really the only, factor of success. The paraffin filling should merely hinder re-expansion of the collapsed lung, but ought never actively to compress the underlying lung tissue. Great compression is responsible for anæmia and consequent sloughing of the tissues, followed by perforation of paraffin into the lung. If performed without misunderstanding

the action of the plug, we can be assured that apicolysis and plombage comply with the one essential principle of collapse therapy. The opinion that the plug rides on the lung and compresses with its weight the underlying tissue is shown to be erroneous at autopsy. We have always found the paraffin plug tightly fastened in a thin fibrous capsule to the thoracic wall, like a tumour of the parietes; it hangs, in fact, in the apical space. This post-mortem finding also shows that a secondary displacement of the plug is nearly impossible. I am inclined to think that this wandering and slipping downwards of the plug (mentioned frequently in the literature) happens only in cases where the appearance of an exudate, soon after operation, enlarges the apicolysis, and by its pressure separates the lung from the thoracic wall. We find then a much larger X-ray shadow than could be accounted for by the quantity of paraffin used. With the disappearance of the exudate the paraffin falls down into the lowest point of the extrapleural cavity created, because in these cases there has been no time for a capsule to form around and anchor the plug.

The one real danger of apicolysis is an accidental tearing into the tuberculous cavity during the operation. Careful technique and experience will nearly always prevent this. Twice it has happened in my series, both patients paying with their lives. In the literature several instances are recorded, though with happy outcome. Sauerbruch recommends suture, if possible ; if not, extensive rib resection and tamponage. Thrice since I have given up an attempted apicolysis, as the separation of the lung from the thoracic wall was so difficult that a similar accident threatened. In such cases rib resection seems more advisable.

On another occasion I created involuntarily an artificial pneumothorax (vainly attempted by the usual method). Although the air-pocket was only very partial, plombage was naturally out of the question. Nevertheless this apicolysis without plombage was followed by a complete success (small apical cavity). Apicolysis without plombage has been advocated by an Italian surgeon as a definite method of collapse therapy, but I fear that, very frequently, the success will not be permanent, as in the case of a pneumothorax persevered in for too short a time. Performed in a more extensive way, such an apicolysis creates an extrapleural pneumothorax. This also has been given a trial—*e.g.*, Nissen, from the Sauerbruch clinic, recommended it for giant cavities; but Sauerbruch himself told me of the complete abandonment of the method.

One important point remains to be mentioned. The posterior route only ensures the possibility of a complete apicolysis. From the second rib, anteriorly, I have never been able to free the whole apex; and have never had, therefore, any permanent good result by this seemingly easier opera-

tion. All these attempted cases had to be completed by a second operation from behind. This is the most important objection to the plombage operation of Winter of Bruges, who uses the pediculated pectoral muscles as plombage material; this naturally removes all objections attaching to the foreign body method, but the apicolysis is probably always insufficient, and has to be completed by a partial thoracoplasty from behind. This latter combination has given really remarkable results to Dr. Winter in cases of giant cavities of the apex, where there can exist no good prospects for paraffin plombage, or even for a simple thoracoplasty.

	Unilateral Plombage, Unilateral Disease.		*Unilateral Plombage, Bilateral Disease.*		*Bilateral Plombage, Bilateral Cavities.*	
	No.	*Per Cent.*	*No.*	*Per Cent.*	*No.*	*Per Cent.*
1. Cured	46	69·7	6	46·2	4	44·4
2. Ameliorated ..	4	6·1	6	46·2	4	44·4
3. Without success ..	14	21·2	1	7·6	—	—
4. Operative mortality	2	3·0	—	—	1	11·1
Total	66	—	13	—	9	—
Durable result 4½ months to 1 year	35	53·0	3	22·8	1	11·1
Secondary mortality ..	14	21·2	1	7·6	1	11·1
(*a*) Of the cured ..	5	10·9	1	7·6	1	11·1
(*b*) Of the non-cured	9	64·3	—	—	—	—

This leads us to the second and, apart from technique, the most important factor of success—*i.e.*, the selection of the suitable case. It is impossible to go into details on this most difficult subject, but as a result of our own experience we are able to make the following remarks : Anatomically those cases are most suitable which have small and medium-sized cavities of the apex; infraclavicular cavities are less so and good results are rare. As regards biological considerations, old, nearly inactive cavities of the fibro-cavernous type with little sputum are best for ensuring immediate and lasting results. In the secondary type of cavity (the usual infraclavicular situation is less favourable) the immediate results have been quite good, but the danger of the very localised collapse not producing a permanent cure is very great, as the immunological state of the patients is frequently not sufficiently high. Relapses below the plombage or in other parts of the lung are therefore frequent, especially if, in addition to the cavity-closing operation, general treatment is neglected. We have spoiled a few anatomically very favourable but biologically unfavourable cases by operating too soon. Thus, in one case, a seemingly small complication

(tonsillitis), and in another a tonsillectomy, shortly after the operation, overcame the unstabilised immunity, and a new spread of the disease, in spite of the closed cavity, was the result. Premature departure from the sanatorium after the operation has also caused quite a number of regrettable and wholly preventable relapses. As the statistics published below might appear to be too favourable, it seems to be doubly my duty to emphasise and insist on the fact that these results have been obtained only in connection with (often prolonged) " cures " at an Alpine sanatorium. Patients with fever and a progressive tuberculosis are not suitable for apicolysis. The eventual closure of the cavity in such cases does not change the course of the disease. To sum up in one sentence: The real indication for the plombage operation is in those cases where the cavity is the one and only obstacle to cure. In such cases a good and permanent result may be anticipated.

CONSULTATIONS

CASE II.

By O. AMREIN,
M.D., F.M.H.,
Medical Superintendent to the former Altein Sanatorium, Arosa, Switzerland.

In 1931 a young man of twenty-four was taken ill with an early infra-clavicular infiltration in his right lung. An artificial pneumothorax was performed at his native town—Frankfurt-Oder. This pneumothorax was maintained until September, 1933. He came out to Arosa at the beginning

Fig. 1.

of February, 1934. He then still showed a small miniature pneumothorax over the upper part of the right lung. The auscultatory and X-ray findings were quite satisfactory. Also his general condition was good. He had a normal temperature, and left Arosa at the end of May, 1934, without any signs of activity. In addition, the sedimentation test of the blood and the differential count of the white blood corpuscles were absolutely normal, but he did not lose a somewhat alarming and very obstinate dry cough, although there was no affection of the throat or higher air-passages.

In May, 1934, not long after his return home, his general condition became worse again, and his physician found several new infiltrative areas at the right lower lobe.

He tried to renew the artificial pneumothorax, and in spite of the formation of thick and hard adhesions, was successful. It remained a small "mantle" pneumothorax, and the refills, though difficult, were continued. In December, 1934, T.B. were found in the sputum. His expectoration had been negative since 1931, and the doctor at home was surprised and grieved to find a new tuberculous process spreading out at the left upper lobe, with the formation of a cavity of the size of a cherry-stone.

FIG. 2.

Now—in January, 1935—the patient was sent again to me for a consultation. After examining him I wrote to the home physician as follows:

"I found the right lung without any alarming symptoms. There is some diminished breathing audible over the lower lobe. But I could not hear any crepitations or râles in the whole of the right lung. The radiograph shows a very small narrow pneumothorax on the right side (mantle); the shadow of the infiltration at the right lower lobe—in comparison with the radiograph taken at Frankfurt two months ago—has cleared up to a certain amount. In the left lung, below the collar-bone, the cavity of the size of a cherry-stone is clearly visible, together with some secondary infiltrative shadows.

"His sputum contains T.B. in small numbers, 4 to 6 per field, also some

elastic fibres. The sedimentation-rate of the red blood corpuscles is too high—*i.e.*, 14 mm. in the average—and there is a deviation to the left in his blood count, the ' stick-formed ' neutrophiles showing 10 per cent. (instead of 3 to 5 per cent.), the ' segmented ' ones 44 per cent. only (against 58 to 66 per cent.) The lymphocytes are increased to 34 per cent.

" I do not doubt one moment that these symptoms of activity, confirmed by blood and sputum tests, are due to the new formation of that cavity at the left upper lobe, whilst, for the present, the right lung seems to be quiescent.

" It is my opinion that a further process of breaking down of lung tissue and the development of a large cavity must be hindered at all costs. I therefore strongly advise that a new artificial pneumothorax ought to be tried on the left side. As the pneumothorax on the right side is a very small one only, and as the refills seem to be very difficult and painful, I recommend that they should be stopped.

" The prognosis remains somewhat doubtful. But we may expect a good effect by collapsing the left lung carefully. As the process there is not of long standing I hardly think adhesions will hinder a good collapse. But of course the crucial point will be whether the right lung will remain quiescent and not show any more alarming signs of activity. One will have to watch very carefully the right lung when performing and continuing the pneumothorax treatment on the left one."*

* The above was written on January 10, 1935. The local doctor and the patient's family wished him to remain here so that I might induce the new pneumothorax myself. This was done without any difficulty on January 20. Up to now (February 27, 1935) the patient feels well, has no temperature, and is spitting hardly at all. The X-ray screening shows a very good collapse of the left upper lobe. The patient is given 300 to 400 c.c. of air with the refills as a maximum. As mentioned above, the condition of the right lung will be the critical question.

CLINICAL CASES

NOTES OF A CASE OF SPONTANEOUS PNEUMOTHORAX

By J. M. TAIT,
M.D.(DUNELM.),
AND
A. S. WAKELY,
M.C., M.B., B.S.(LOND.),

H. N., æt. 32, was apparently a healthy man of medium build leading a quiet life in a sedentary occupation. He took no physical exercise except walking and a certain amount of lifting in connection with an invalid relative. His only previous illness in the last ten years was influenza ten years ago.

History.

While engaged in moving from one house to another, he lifted the end of a bed and, fifteen minutes later, became conscious of a pain in the right side of his chest.

Second Day.—He went to his office feeling slightly short of breath.

Third Day.—He was seen by one of us. He was up and dressed and not distressed. He complained of pain in the right side of his chest on deep inspiration and on exertion. On examination his temperature was normal. Pulse-rate 90. Respirations 20. His chest showed signs of a right-sided pneumothorax.

Fourth Day.—In spite of complete rest in bed he was dyspnœic and had a larger amount of air in the pleural cavity. Temperature 101°. Pulse 100. Respirations 32.

Fifth Day.—His condition became more urgent, with marked respiratory distress and cardiac displacement. The pressure was reduced at 4 p.m. with some relief. An urgent call seven hours later was occasioned by his complete collapse. Respirations were slow and laboured. The pulse was about 160, thready and intermittent. He was pale and clammy. The right side of his chest was dull to percussion up to the level of the fifth rib,

above which the hyperresonant note of the pneumothorax persisted. The pleural cavity was tapped in the third interspace, from which air escaped under considerable pressure, with immediate relief to the distressing symptoms. Before the pressure was sufficiently diminished, however, pure blood began to escape. This prevented us from carrying out our intention of leaving the needle with pressure adjustment *in situ*. He reacted to this by an increase in the respiration-rate; the breathing became much less difficult and his pulse improved. From semi-consciousness his mind became clear, and he stated he was comfortable and felt much better. On account of the large intrapleural hæmorrhage that had occurred, antishock treatment was given, including continuous saline, hæmoplastin, and coramine. Heroin ½ grain gave him a good night.

Sixth Day.—His general condition was still poor. He had a cough with blood-stained sputum. The temperature remained at 100°. Dyspnœa increased during the day, and dullness on the right side had by now risen to the level of the third rib. In the evening a further puncture was made well above the level of the fluid. Air escaped in much smaller quantity, but afforded some relief. He had a restless night, with cough and some delirium at times.

Seventh Day.—The general condition was slightly improved. The right side was dull except for resonance in the first and second interspaces. Temperature 100°. Pulse 116 and weak. Respirations 40.

Eighth Day.—Sudden collapse and death in the early morning.

Post-Mortem.

The following facts were noted:

On opening the chest, a small amount of air escaped from the right pleural cavity. The right lung was completely collapsed. The right pleural cavity was filled by 2½ pints of blood and a large amount of recent blood clot. The upper surface of the diaphragm and portion of the upper surface of the lung showed deposits of fibrin—(?) organisation of blood clot.

The left pleural cavity showed nothing abnormal apart from a small amount of free fluid.

Right Lung.—Complete collapse. At the apex and for a short distance along the anterior border there were a series of bullæ of varying sizes about twelve in number ranging, when distended, up to the size of a small cherry. One of these, near the apex of the lung, had perforated; the aperture was filled with recent blood.

Left Lung.—This showed a similar condition apart from the collapse. The bullæ on this side were even larger and more numerous, one in particular being elongated to the size and shape of a date-stone.

There were no pleural adhesions nor signs of pleurisy at either apex. The bronchi of both sides contained a small amount of purulent material. There was no evidence of tuberculous infiltration in either lung. The bronchial glands were not enlarged.

Heart.—No enlargement. The heart muscle and valves appeared healthy.

Our thanks are due to Dr. A. Hope Gosse for advice regarding treatment and for encouragement and help in recording this case.

CHRONIC IDIOPATHIC SPONTANEOUS PNEUMOTHORAX

By J. W. CRAIG,
M.D.(DUBLIN).

I DO not intend to discuss the whole subject of pneumothorax, and will merely record this case and mention the literature which has some bearing on the subject. The case was in the Brompton Hospital under the care of Dr. Burrell, to whom I am indebted for kind permission to publish these notes.

Hydro-pneumothorax was recognised by Hippocrates, but it was not until the nineteenth century that pneumothorax was described as a complication of pulmonary tuberculosis by Laennec and Itard. Saussaur[1] in 1841 collected 147 cases occurring without any diagnosable underlying lesion of the lung, and in 1887 Hall and West[2] described the sudden onset of pneumothorax in healthy people whilst resting or sleeping.

Many cases have since been recorded, and like other rare conditions this is met with more often than is sometimes realised.

The pathological conditions causing pneumothorax are numerous. Laennec,[3] Saussaur, West, etc., have discussed the pathological anatomy. All place tuberculosis at the head of the list of causes. Some writers—for example, Weber[4]—believe that all pneumothoraces which cannot be explained on the basis of some other morbid change are due to rupture of an area of healed tuberculosis.

Pneumonia, empyema, lung abscess, pulmonary gangrene, bronchiectasis, emphysema and asthma, pulmonary infarct and hydatid cyst, have all been recognised as the primary underlying condition.

Cases without such definite cause are classified as " idiopathic," and the usual explanation is that of rupture of a small emphysematous bulla, or tear of an adhesion.

The etiology of " idiopathic " spontaneous pneumothorax is speculative, and le Wald,[5] who reported ten cases of his own, found that the condition occurred in perfectly healthy people who subsequently made a complete recovery. He, however, noticed cases persisting for years. In the discussion which followed his paper many agreed that all pneumothoraces of unknown origin should be regarded as tuberculous.

Leggett, Myers, and Levine,[6] who have made an exhaustive study of this subject, concluded that though many doubtful cases are tuberculous in origin, they should not all be classified as such.

They noticed that idiopathic spontaneous pneumothorax in apparently healthy individuals was usually followed by re-expansion of the collapsed lung and clinical recovery. Vrooman[7] came to the same conclusion. The case we are reporting is unusual in that the lung is still completely collapsed after one year's duration.

Other cases of chronic idiopathic pneumothorax have been reported, but are unusual. le Wald[5] has published the notes of a case lasting ten years, and of another in which the lung has remained collapsed for over one year.

Muggia[8] has recorded the interesting fact of a spontaneous pneumothorax persisting in a child for three years. This was subsequently cured during an attack of whooping-cough. Bryce[9] described two cases of chronic pneumothorax, but these had a history which suggested tuberculosis.

In the case here reported, the absence of cough, of pyrexia and of pleural fluid, the X-ray and absence of abnormal physical signs in the better lung, the negative results of examination of the sputum for tubercle bacilli, the normal blood sedimentation-rate, and the thoracoscopic findings suggest that tuberculosis was not the cause of the pneumothorax, which has lasted for one year.

Female, aged thirty-five years. No family history of pulmonary tuberculosis. Past history: Enteric fever, 1911. Otherwise always in good health, but not very robust. Has had trouble with maxillary antrum.

March, 1934, onset of sudden pain in right side of chest and felt breathless. Advised to go for a holiday; fourteen days later had a second attack of pain and felt very dyspnœic.

In June, 1934, she had her antrum washed out, and as she complained of her chest discomfort the doctor made an X-ray examination and found a partial right pneumothorax. Three days later, when in a nursing home, she had severe pain and felt very breathless. It was discovered that the pneumothorax had greatly increased. A pneumothorax needle was inserted and some air removed.

November 30, 1934: Streaky hæmoptysis.

December 3, 1934, admitted to Brompton Hospital under Dr. Burrell. General condition good. Chest: No breath sounds on the right side, heart to the left. Blood pressure 145·80. Blood sedimentation normal.

Symptoms.—Very slight cough. Trace of sputum, but no tubercle bacilli. Slightly streaky hæmoptysis. Dyspnœa on exertion.

X-ray showed the right lung completely collapsed and the heart to the left. There was no evidence of pulmonary tuberculosis.

An effort was then made to expand the lung in the hope that it might adhere to the chest wall and so close the opening.

December 5, 1934: A needle was inserted and the pressure found to be +6 +2. 1,400 c.c. air withdrawn and the pressure settled to −6 −3.

December 6, 1934: Pressure −2 +3. 1,900 c.c. air withdrawn, and pressure −10 −2.

December 10, 1934: Pressure −4 −2. 1,600 c.c. air withdrawn. X-ray showed normal-looking lung considerably re-expanded.

December 11, 1934: Screened. Lung collapsed again.

December 12, 1934: 2,415 c.c. air drawn slowly off with drop syphon bottle; initial pressure −4 −0. Final −9 −4.

December 13, 1934: Pressure −2 +4. Lung collapsed.

December 21, 1934: Under water-drain for two days. Continual negative pressure, but air bubbled through all the time. Lung slightly re-expanded.

January 18, 1935: Thoracoscopy. Lung completely collapsed; only abnormality discovered was a small dimple in postero-lateral angle of lower lobe. Red spot in this depression. No opening seen. At the apex a band-like adhesion was seen. No tubercles.

February 1, 1935: Patient fairly comfortable, and it has been decided to let her go home and rest. Operation did not seem justifiable, and it is hoped that the valvular opening will close and the lung re-expand in time.

REFERENCES

1. Saussaur: *Thèse*, Paris, 1841.
2. S. West: *Lancet*, May 3, 1884.
3. R. T. H. Laennec: *De l'Auscultation Mediate*, 1819.
4. F. P. Weber: *Brit. Med. Journ.*, 1931, ii. 989.
5. L. T. le Wald: *Arch. Surgery*, 1928, xvi. 426.
6. Leggett, Myers and Levine: *Am. Rev. Tuber.*, March, 1934, pp. 348-361.
7. C. H. Vrooman: *Canadian M.A.J.*, March, 1934, xxx. 265-268.
8. R. Muggia: *Renasc. Med.*, February 28, 1934, ii. 109-110.
9. A. G. Bryce: *Brit. Med. Journ.*, April 4, 1934, i. 714.

MEETINGS OF SOCIETIES

THE TUBERCULOSIS ASSOCIATION

THE January meeting of the Association was held at Manson House, W. 1, under the chairmanship of the President, Dr. L. S. T. Burrell.

At the afternoon session Dr. P. F. Armand-Delille read a paper on "Pulmonary Tuberculosis in Adolescence and Youth." He began by pointing out that the old conception that pulmonary tuberculosis in adolescents was invariably the sequel to repeated contact with tuberculosis germ carriers had given way to the modern theory, now universally held, that the development of pulmonary tuberculosis in early adolescence practically always meant local reinfection of an organism which had received its primary infection in infancy. Reinfection was probably usually autogenous, and not exogenous, as was formerly believed. X-ray films showed that miliary tuberculosis was by no means always fatal. Many cases of the miliary condition known as "granulie froide" had been observed in childhood and in adolescence. The frequent occurrence of tuberculosis among young nurses or students in tuberculous wards or clinics might, the speaker thought, be accounted for by the suggestion that absorption of bacilli by contact with germ carriers did not actually provoke reinfection, but determined, like a tuberculin injection, the reactivation of latent foci.

There were three main pathological types of pulmonary tuberculosis in adolescents: (1) Spleno-pneumonia, first described by Graucher in 1880, and known more recently as epituberculosis or perifocal reaction; (2) the pneumonic form; and (3) chronic fibro-ulcero-caseous lobitis. *Spleno-pneumonia* was characterised by marked congestive reaction, indicating that the allergy was not fully developed. It was common in young children, and sometimes appeared after an acute infection, such as measles. It always began with a typhoid type of fever, followed by the symptoms of pleurisy, but the absence of fluid on puncture and a somewhat characteristic X-ray shadow should serve to differentiate it. The course of the disease was regressive, and except in cases of accidental death the pathology of the condition could not be studied. It was often associated with erythema nodosum, which was also a manifestation of hypersensitiveness and incomplete allergy.

The *pneumonic* type of tuberculous disease was rarely seen post-mortem in its early stage, because as a rule after the first condensation or hepatisation it produced a cavity, and at death the aspect was that of chronic tuberculosis of ulcero-caseous type. If these cases were not collapsed by artificial pneumothorax, the condition would go on to the classical fibro-ulcero-caseous involvement of the whole lung. Clinically, this type of case began rather suddenly, the first diagnosis being usually acute pneumococcus pneumonia. The symptoms and clinical course, however, differed in some respects, and there was no crisis. At the end of the second week tubercle bacilli could usually be found on gastric lavage. Characteristic cavity formation was the next stage, and unless collapse therapy was started immediately, the disease would progress very rapidly—a true "galloping tuberculosis"—death occurring in a few months. In favourable cases with an early induction of pneumothorax the lesions might heal, although sometimes a double pneumothorax was required.

The *fibro-ulcerous* type, as in the pneumonic type, could not usually be studied post-mortem in its early stage. When death did at length occur it would be found, as a rule, that the lobe first attacked had been transformed into a mass of tuberculous tissue, half caseous, half fibrous, and in the other lobe there would be terminal miliary or more or less advanced caseous lesions. Clinically, this form was more insidious and therefore often more dangerous than the other types, and it was often too late when the child was brought to the out-patient department for general examination. If the disease was detected early enough and pneumothorax performed while the lesion was still localised, recovery would as a rule take place. If neglected or inadequately treated, a rapid extension was certain, terminating in death.

In practice, these three principal types of lung tuberculosis in late childhood were not always clear cut, for there were many intermediate forms. Yet one more type, *tuberculose incipiens*, known to German workers as "infraclavicular infiltration," or "Früh Infiltrat," was of some importance. There were no characteristic symptoms in such cases, and they were discovered as a rule only by routine fluoroscopy and radiography.

With regard to treatment, Dr. Armand-Delille thought that no medical treatment had really any effect. Patients given gold treatment did no better than others without it. The only really effective treatment was artificial pneumothorax, started at a comparatively early stage and kept up for a sufficiently long period—say an average of three years.

A number of excellent specimens and photographs were shown, and a vote of thanks was passed to Dr. Armand-Delille for his interesting paper.

At the evening session Dr. J. W. S. Blacklock read a paper entitled

" A Study of Tuberculous Disease in Infancy and Childhood, with Particular Reference to the Primary Sites of Infection." This we have published in full in this number.

The next paper, entitled " The Development of Pulmonary Tuberculosis in Children," was then read by Dr. A. G. L. Reade, who said he proposed to describe the four main types of case admitted into High Wood Hospital for Children, and to show X-ray pictures illustrative of each type. The first he would call the adult type, seen most frequently in the young adolescent who might have left school and just started work. The history often seemed astonishingly short, and the prognosis as a rule was bad. Another type was that of a widespread mottling over both lungs, suggesting miliary tuberculosis. These cases usually ended fatally, but the course was often definitely longer than was usual in acute miliary tuberculosis. About one-fifth of the admissions for pulmonary tuberculosis belonged to the third type, that of pleural effusion. These cases, when uncomplicated, had a very good prognosis, immediate and ultimate, as only a very small proportion returned later for treatment. The fourth group, to which the largest proportion of admissions belonged, were those sent either for diagnosis, or having been notified before admission with such doubtful diagnoses as epituberculosis, hilar type of pulmonary tuberculosis, enlarged hilum glands, or tuberculous mediastinal or bronchial glands. Children suffering from cough, malnutrition, debility, and so on, used to be labelled tuberculous if X-ray examination showed any definite shadows at the roots of the lung. Now it was generally accepted that most of these shadows were not tuberculous at all. Possibly the pendulum had swung too far in this direction; at any rate, it was important to ascertain whether or not the cases were really tuberculous, and, if they were, what exactly these hilum shadows represented. They endeavoured at High Wood to decide the first point by the routine Mantoux test, gastric lavage, lipiodol examination in certain cases, and lateral as well as antero-posterior X-ray examination. Dr. Reade showed a number of radiograms illustrating the different types of hilar shadows, and after a short discussion the meeting closed.

THE TUBERCULOSIS ASSOCIATION

A MEETING of the Association was held on Friday, February 15, the President, Dr. L. S. T. Burrell, being in the Chair. The afternoon session opened with a paper on " Tuberculin," by Dr. Halliday Sutherland. He began by referring to the value of the tuberculin tests in the diagnosis of pulmonary tuberculosis. For the subcutaneous test the patient's temperature must

be normal and recorded at 8 a.m., 4 p.m., and 8 p.m. daily. The amounts of tuberculin injected were: 0·0001 c.c., 0·0002 c.c., 0·001 c.c., 0·005 c.c., 0·01 c.c., the injections being given at intervals of four days. A rise of temperature meant a positive reaction. If the temperature rose half a degree F. the exciting dose should be increased, so that a more definite febrile reaction might be obtained. Once the febrile reaction occurred the test was discontinued. A febrile reaction to 0·0001 c.c., 0·0002 c.c., 0·001 c.c. was diagnostic of tuberculosis. A febrile reaction to 0·005 c.c. was more doubtful, and no one would diagnose active disease from a febrile reaction to 0·01 c.c. On the other hand, no reaction to 0·01 c.c. excluded the possibility of active tuberculosis. In the case of cattle, Kolmer analysed the results of 15,000 injections. Post-mortem examination of the animals showed that the error in the subcutaneous test was less than 3 per cent. The other tuberculin tests in use were the cutaneous and the intracutaneous tests. The latter was the more delicate, and if there was no reaction to an injection of 0·1 c.c. of a one in 10,000 solution, a further injection of 0·1 c.c. of one in 1,000 solution might be given after an interval of four days. It was sometimes said that the subcutaneous test was dangerous, but the first dose in the subcutaneous test—that is, 0·0001 c.c. of Old Tuberculin—was exactly the same amount as was frequently given in the intracutaneous test. In all these tests some tuberculin was absorbed into the system, and the only difference between the subcutaneous test and the intracutaneous test was that in the latter the tuberculin was more slowly absorbed. D'Arcy Hart had noted systemic and focal reactions after the intracutaneous test.

In conclusion, he repeated his regret that the subcutaneous test was not more frequently employed, and the simpler cutaneous test universally employed in the case of young children.

Dr. W. H. Tytler then read a paper on " The Nature of Tuberculin." He said that during recent years there had been a definite advance in their knowledge of the chemical side of the problem, and every technical advance offered new suggestions for approach to the deeper problem—the isolation and identification of the actual chemical entity with which activity was associated, leading to the study of the mechanism of its activity. It was still uncertain to what extent the tuberculin reaction could be elucidated by the chemical study of tuberculin, but the work of recent years had indicated almost certainly that tuberculin activity was associated solely with the protein fraction of the tubercle bacillus. An important point to remember was that a given protein and its degeneration products might form a series of substances of widely varying molecular size, all, however, until the lower members were reached, having similar chemical structure and similar

potential chemical activity. The biological activity might, however, differ throughout the series, the differences in molecular size causing differences in physical behaviour which might decide whether or not the chemical activity could come into play. Tuberculin activity may be exhibited by a range of molecules of varying size, all of which appeared to be derived from the natural large molecular protein fraction of the bacillus. This fraction probably contained at least two distinct proteins, and though the various derivatives might have different biological effects, the speaker thought the differences could be traced to differences in molecular size alone. Two sources were available for the preparation of active tuberculin fractions: the tubercle bacillus growth itself and the fluid culture media on which it had grown. The aqueous or alkaline extracts of tubercle bacilli had high tuberculin activity, the active material being almost entirely in the form of large molecular protein of two types: a water-soluble nucleo-protein and a phosphorus-free alkali-soluble fraction. This protein produced anaphylaxis and antibody formation; it was highly active for the intradermal tuberculin reaction, but had a much lower capacity for producing a general toxic or lethal reaction. In the unheated culture fluid about one-quarter of the tuberculin activity was due to large molecular protein, mostly resembling the alkali-soluble type. The bulk of the activity in the culture fluid, however, was due to small molecular material, possibly below the limits of protein size. It is not precipitated by acetic acid, but was mostly brought down by trichloracetic acid, and this precipitate had high skin reaction activity, but did not sensitise or cause antibody formation. There was also present material definitely below the protein level, which had little power to elicit a skin reaction, but which was actively toxic or lethal for tuberculous animals. Coming to the question of the most satisfactory product for diagnostic testing, the American workers had come back in a roundabout way to active material in the precise form in which it was present in Old Tuberculin, as being free from the risk of sensitisation. The greatest drawback to Old Tuberculin was the wide variation in activity between different batches and the inexact methods of standardisation. The most obvious advantages of the purified fractions were that they were indefinitely stable in dry form, and that they were exceedingly constant in activity. The unequal distribution of skin reacting and lethal material present in Old Tuberculin raised a practical point in its use. Two tuberculins which had equal value when standardised by the intradermal method would not necessarily be equivalent on subcutaneous injection, and *vice versa*. This difficulty would, of course, be obviated by the use of a single fraction, or, better, of an appropriate fraction for each method.

Finally, Dr. Tytler emphasised once more the point that the development

of a diagnostic reagent was only of secondary importance as compared with the problem of explaining the tuberculin reaction, but the tracing of activity down through progressively smaller molecules did seem to bring them nearer to the possibility of identifying a definite chemical entity as the essential active principle of tuberculin.

At the evening meeting Dr. G. Gregory Kayne (Dorothy Temple Cross Research Fellow in Tuberculosis) read a paper on B.C.G. vaccination in Western Europe. Dealing first with the question of virulence, Dr. Kayne said that recent work with the tubercle bacillus had shown the difficulty of fixing a clear line of demarcation between virulence and avirulence. Living tubercle bacilli could be broadly classified into four groups, producing increasingly milder effects: progressive tuberculous disease; tuberculous lesions spontaneously regressive; lesions not typically tuberculous, but which nevertheless produced some tuberculin hypersensitiveness and immunity; and, lastly, minimal or no lesions, unassociated with the last two phenomena. Bacilli in the first two groups might be termed virulent, and the lesions were reinoculable in animals. B.C.G. was claimed to represent the third group, and three questions had to be answered: (1) Did B.C.G. never cause progressive or even spontaneously regressive tuberculous lesions in animals or man? (2) Could B.C.G., however grown, increase in virulence so as to produce progressive tuberculosis? (3) Did B.C.G., when grown as recommended by Calmette, remain absolutely unchanged—*i.e.*, neither increasing in virulence nor becoming more attenuated? With regard to the first of these questions, the speaker said that he had found no evidence of a progressive lesion being produced in animals or man in any of the seven European countries visited by him. There was also a general agreement that there had occurred no increase in the original virulence of B.C.G. as grown according to Calmette's latest instructions; but even if by subculturing B.C.G. in certain other ways a return in virulence was a possibility, as stated by various workers, Dr. Kayne pointed out that in any case such a possibility was no contra-indication to the application of the vaccination in man, provided the routine method of subculture did not have that effect. On the other hand, experiments *in vitro* and *in vivo*, as well as clinical evidence, indicated a definite attenuation of the Pasteur Institute strain in recent years. B.C.G. was not a *virus fixe*. Grown on media like glycerine-potato, it very slowly became further attenuated. Grown on bile-potato only, it appeared not to become attenuated, and perhaps even increased slightly in virulence. Although B.C.G. was, he believed, definitely innocuous in man, the B.C.G. used at present did appear to vary in virulence in different countries. For its application in man over a number of years it was essential

to find a method of culture which would not attenuate it to the stage when little or no tuberculin hypersensitiveness, and therefore presumably immunity, was produced, or alternatively a method must be found of slightly raising its virulence when it did so. This problem was being tackled at the State Serum Institute at Copenhagen.

Before any conclusion could be drawn as to the immunity conferred by B.C.G., it was essential that the vaccine should have entered the tissues and have reacted with them. This implied that (*a*) all the bacilli in the vaccine administered should be alive; (*b*) that all the bacilli in the vaccine administered should gain entrance to the tissues, so that the effective dose in each case could be known—this being only possible in the parenteral methods; and (*c*) a positive tuberculin test should be obtained. Provided the child had not in the interval been in contact with tuberculosis, this last condition was the only indication that the vaccine had reacted with the tissues. The nature of the tuberculin test was of obvious importance. Except for young children, for whom the Pirquet test might suffice, the Mantoux test carried out to 1 mgm. should always be used for accurate work. The determination of immunity produced by vaccination could in man only be made by considering the tuberculosis morbidity and mortality in vaccinated subjects. Exact controls were extremely difficult to obtain, and the speaker criticised in some detail many of the published statistical investigations. Analysis of the evidence suggested that B.C.G. produced a very partial immunity which was only likely to play an effective rôle when it was combined with other methods of prophylaxis; and that the duration of the immunity varied considerably, but was generally between six and twelve months.

JOINT TUBERCULOSIS COUNCIL

At the February meeting of Council twenty-six members were present. Dr. Lissant Cox was elected Chairman for 1935, Drs. L. S. T. Burrell and S. Vere Pearson Vice-Chairmen, Dr. Ernest Ward Hon. Secretary, Dr. G. Jessel Hon. Treasurer, and Dr. D. P. Sutherland Hon. Auditor.

Drs. J. B. McDougall and W. H. Tytler were re-elected, their term of membership having expired.

The Hon. Secretary gave a report on the proceedings for 1934. There had been four meetings, one of them a special meeting, with an average attendance of 21·5, in spite of the fact that only thirteen attended the special meeting. The Council's productions were still in demand from all over the world—Hungary had recently asked for them. The most sought after in 1934 were that by Dr. W. H. Tytler on Tuberculin, and the Memo. of

the Institutions Committee, of which Dr. Blackmore is convener. The Medical Research Council had granted £200 towards the expenses of the committee investigating the after-results of pneumothorax treatment, of which Dr. R. R. Trail is the convener.

The special meeting was an innovation and had established certain precedents as to the convening of such meetings. Those desiring a special meeting should prepare and sign a memo. giving their reasons, and to serve as a basis for discussion. This would be considered by the officers of the Society and their decision passed to the Secretary for appropriate action. Another precedent established in 1934 was the co-option of Dr. Dawkins, a non-member, on the Contacts Committee.

The Hon. Treasurer presented his report for 1934. The expenses had been rather higher, owing mainly to the printing and circulation of various literature.

Professor Jameson explained the note which he had circulated on behalf of the Milk Committee, dealing with attested herds. Producers feared great difficulties in working the scheme. Dr. Hawthorne drew attention to two circulars issued by the Ministry of Agriculture and Fisheries. Circular A920 TA advocated safe milk, and a circular dated January, 1935, advised that milk used for animal feeding should also be safe milk. A very definite advance in policy.

Dr. Ernest Ward presented a note on the Prevention of the Common Cold and on Spahlinger Treatment. For these he was thanked.

Concerning post-graduate study, Dr. Brand announced further details of the courses to be given by Dr. Peter Edwards, and of the course at Victoria Park Chest Hospital. He also outlined a most attractive study course in Denmark to take place in June. The Secretary raised the question of intensive post-graduate courses for nurses, and various members expressed belief that there was a general demand for such courses. Drs. Blackmore, Jessel, and Esther Carling were asked to discuss the question with the Examination Committee of the Tuberculosis Association at their April meeting, and Dr. Ward was asked to get in touch with the General Nursing Council and the College of Nursing there-anent.

As regards future work, Dr. Heaf promised to prepare a note on " Tuberculosis Institutions and Chest Hospitals," and Dr. Tytler was asked for a note on " The Examination of Sputum for Tubercle Bacilli."

The standardisation of the technique of blood sedimentation tests was raised by Dr. Hebert, and it was decided to place this subject on the agenda for the next meeting.

NOTES AND NEWS

THE Post-Graduate Courses are arranged by Dr. William Brand on behalf of the Joint Tuberculosis Council.

In February the Medical and Surgical Staff of the City of London Hospital for Diseases of the Heart and Lungs gave a six-day course which was most successful.

In March, Dr. Peter Edwards gave an intensive course at the Cheshire Joint Sanatorium. Dr. Peter Edwards will hold a similar course from May 7 to 9, and again from June 4 to 6. These courses deal with modern methods of treatment in pulmonary tuberculosis, with special reference to collapse therapy. The fee for the course is £2 7s., which includes lunch and tea at the sanatorium.

Arrangements are also being made for a course to be held in Denmark in June. Those wishing to attend should apply early to Dr. Brand.

In the autumn a course is to be held at the Brompton Hospital, details of which will be given later.

POST-GRADUATE TEACHING AT THE BROMPTON HOSPITAL

The following arrangements have been made for post-graduates wishing to undertake a course of special work at this hospital:

(1) Clinical Assistants. These are appointed for a period of three months in the wards and out-patient department on the medical and surgical sides. Duties are as far as possible whole-time, but special arrangements can be made if necessary. Fee: £5 5s.

(2) The "Practice" of the Hospital. Fee: £1 1s. for each month. It is hoped that those taking up this course will spend as much time as possible in the wards, out-patient, and special departments.

(3) Post-graduates desiring to attach themselves to one or other of the special departments for a short period can do so by special arrangements, the terms for which can be obtained on application to the Dean.

(4) Two Post-Graduate Courses of one week's duration each and three week-end courses are held during the year.

(5) There are in addition classes for candidates for the M.R.C.P. These are held twice a week for one month before each examination.

(6) Under-Graduate Classes are also held twice a week for one month before the final examinations.

With the exception of the Under-Graduate Demonstrations, these classes and appointments are held in connection with the Post-Graduate Association, to whom application in the first instance should be made.

REVIEWS OF NEW BOOKS

Averages of Bright Sunshine for the British Isles for Periods Ending 1930. Published by the authority of the Meteorological Committee, Air Ministry, M.O. 377. Price 1s. H.M. Stationery Office.

This publication contains two main tables, the first of which gives average monthly totals, daily means and percentages of possible duration of sunshine for all stations having ten years or more of observations in the period 1901-1930. The second table gives for 18 selected stations the average and percentage number of days in each month, with sunshine within stated limits of duration.

The total number of stations for which averages are printed is 172, of which 31 are in Scotland and 9 in Ireland.

The data show that the average annual duration of recorded sunshine varies from over 1,800 hours in the Channel Isles and on the South Coast of England to under 1,100 hours in the Shetlands and in certain industrial areas where heavy smoke pollution occurs. At all stations May or June is the sunniest month, and December or January the dullest.

A Practical Manual of Diseases of the Chest. By MAURICE DAVIDSON, M.D., F.R.C.P., Physician to the Brompton Hospital for Consumption and Diseases of the Chest and to the Miller General Hospital. Pp. 528. 1935. Price £2 2s. Oxford University Press.

This work, though primarily intended for those who specialise in diseases of the chest, is not a general treatise, but a book of moderate size unburdened with too much detail.

With reference to tuberculous subjects the reader will find a good account of the treatment of pleural effusion. The probability of the condition being tuberculous and the dangers of early aspiration are emphasised, and reports of 4 cases are given at the end of the chapter.

One hundred and twenty-three pages are devoted to pulmonary tuberculosis, and give a very concise résumé of the modern views of this complicated disease. During the last twenty-five years there have been vast changes not only in our conception, but in our treatment of pulmonary tuberculosis. Masterly inactivity of a quarter of a century ago has been replaced by such activity that even the removal of eleven ribs is now regarded as part only of the cure.

Dr. Davidson has steered a middle course, and whilst doing full justice to modern ideas, has written with restraint not only as regards treatment, but also as regards various tests and methods of prevention.

Diagnosis is a matter of great difficulty, and attention should specially

be drawn to Chapter XXIII., in which certain obscure clinical and radiological conditions are discussed, including epituberculosis and acute benign pulmonary tuberculosis, and reports of several cases are given.

The book is very well printed and produced, and the numerous radiograms are really excellent. It is a work for which there is a real demand and one that can be warmly recommended.

Clinical Methods. (Tenth edition.) By ROBERT HUTCHISON, M.D. Edin., F.R.C.P. Lond.; and DONALD HUNTER, M.D., F.R.C.P. Lond. Pp. 616, with 21 plates and 142 text-figures. Price 12s. 6d. net. Cassell and Co., Ltd., La Belle Sauvage, London, E.C. 4.

This well-known book, which was first published in 1897, has been reprinted thirty-four times, so great is its popularity.

In this edition the book has been thoroughly revised, but the chief changes will be found in the chapters dealing with the examination of the blood, urine, and nervous system.

One chapter is devoted to a full description of the examination of the respiratory system, and the classical physical signs are explained. There is no agreement amongst clinicians as to the exact meaning of such terms as "râles," "crepitations," "bronchial breathing," "tubular breathing," and consequently there is a great deal of confusion amongst students. We agree with the authors that the term "râle" might well be dropped, but not if, as they state, fine crepitations (which characteristically are heard when the alveoli are full of exudate in pneumonia) are due to the opening-up of closed alveoli and not to minute bubbles of air in the secretion. Vesicular breathing is divided into six varieties and bronchial breathing into two each, with three sub-divisions. The expert as well as the student will find it difficult to know what is meant by "medium-pitched bronchial" breathing and by what pulmonary condition it is produced. It is true that one type of breathing gradually merges into another as day merges into night, but just as we speak of light, twilight, and dark, so we might find some simpler nomenclature for breath sounds.

The part which gives an account of the physical signs of the principal pulmonary diseases might be omitted in the next edition. The twenty-one lines devoted to pulmonary tuberculosis can be but of small value to the student.

The first chapter on case-taking is excellent, and teaches the student to be thorough, not only in physical examination, but also in the all-important task of taking a good history.

No student can be without this little book, and the authors are to be congratulated on adding so much new material regarding clinical and laboratory methods of examination and at the same time keeping the same conveniently small size of the book.